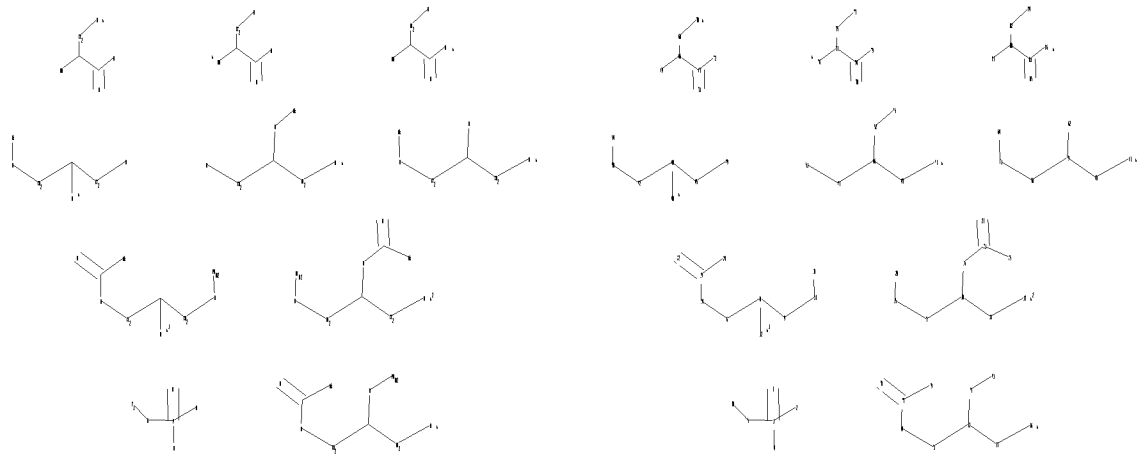
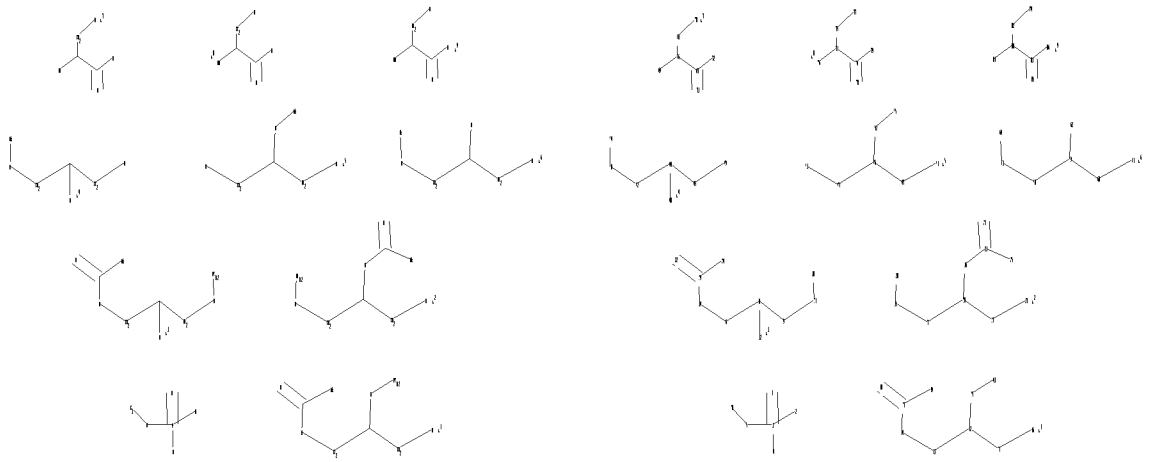
```
     FILE 'REGISTRY' ENTERED AT 10:33:28 ON 01 OCT 2009
L1                 STRUCTURE UPLOADED
L2               0 S L1
L3                 STRUCTURE UPLOADED
L4               0 S L3
L5                 STRUCTURE UPLOADED
L6               0 S L5
L7               2 S L5 SSS FULL
L8                 STRUCTURE UPLOADED
L9               0 S L8
L10              0 S L8 SSS FULL

     FILE 'STNGUIDE' ENTERED AT 10:38:12 ON 01 OCT 2009

     FILE 'HCAPLUS' ENTERED AT 10:43:33 ON 01 OCT 2009
L11              0 S (LYSOPHOSPHTIDIC ACID)(4A)(INHIB? OR ANTAG?)
L12         717126 S PHOSPHATE OR PHROPHOSPHATE OR PHOSPHORIC
L13         220274 S CHOLESTEROL OR HYPERCHOLESTEROL? OR HYPERLIPID? OR ATEHROSCLE
L14              0 S L11 AND L12
L15              0 S L11 AND L13
L16           6976 S L12 AND L13

     FILE 'STNGUIDE' ENTERED AT 10:43:40 ON 01 OCT 2009

     FILE 'HCAPLUS' ENTERED AT 10:44:18 ON 01 OCT 2009
L17            314 S (LYSOPHOSPHATIDIC ACID)(4A)(INHIB? OR ANTAG?)
L18         744163 S PHOSPHATE OR PYROPHOSPHATE OR PHOSPHORIC
L19         220274 S CHOLESTEROL OR HYPERCHOLESTEROL? OR HYPERLIPID? OR ATEHROSCLE
L20             62 S L17 AND L18
L21              3 S L17 AND L19
L22           7389 S L18 AND L19

     FILE 'REGISTRY' ENTERED AT 15:38:02 ON 01 OCT 2009
                   EXP SERINE PHOS/CN
                   EXP SERINE PHOSPHATE/CN
L1                1 S E5
                   EXP SERINE PHOSPHORIC/CN

     FILE 'HCAPLUS' ENTERED AT 15:38:45 ON 01 OCT 2009
L2               67 S L1/THU
L3           194608 S NEOINTIM? OR ATHEROSCLEROSIS OR STENT OR CARDIOVASCULAR
L4               0 S L2 AND L3
L5              31 S L2 AND (PY<2003 OR AY<2003 OR PRY<2003)
```

```
=> file registry
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.22       0.22

FILE 'REGISTRY' ENTERED AT 10:33:28 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2009 American Chemical Society (ACS)

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:   29 SEP 2009  HIGHEST RN 1186580-18-6
DICTIONARY FILE UPDATES:  29 SEP 2009  HIGHEST RN 1186580-18-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=>
Uploading C:\Program Files\STNEXP\Queries\10821739monophosphate.str
```



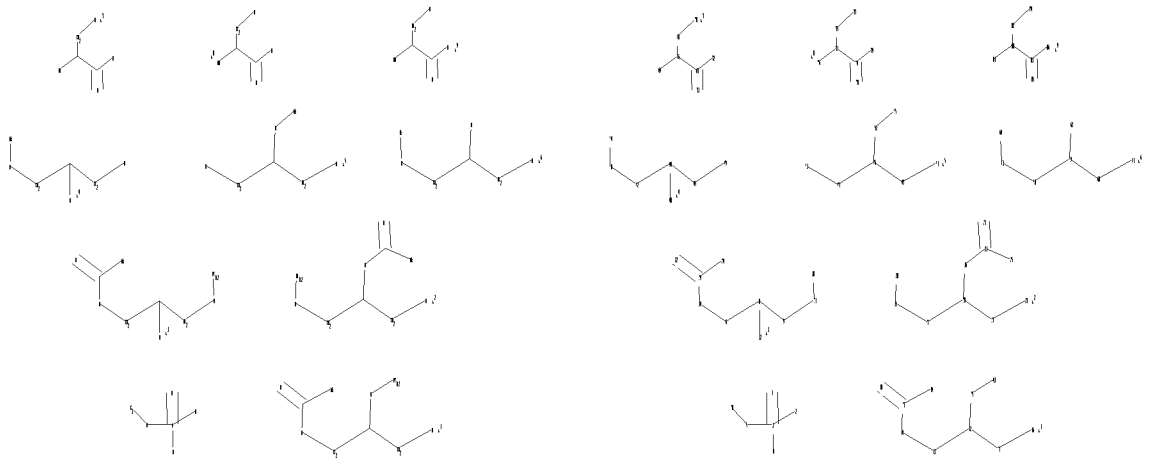
chain nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
24 25 30 31 32 33 34 35 36 37 38 39 41 42 43 44 45 46 47 48 49
50 51 52 53
54 55 58 59 60 61 62 63 64 66 67 68 69 70 71 72 73 74 75 76 77
78 79 80
81 82 83 84 85 86
chain bonds :
1-2 1-3 1-4 1-5 5-30 6-8 6-14 7-10 7-15 8-9 8-12 9-13 10-11 10-16
11-17
13-18 14-19 15-20 16-21 19-22 19-24 21-23 21-25 31-32 31-34 32-33 32-35
33-36 34-37
35-41 37-38 37-39 42-44 42-50 43-46 43-51 44-45 44-48 45-49 46-47 46-52
47-53 50-54
52-55 58-59 58-61 59-60 59-62 60-63 61-64 66-67 66-68 66-69 67-71 67-72
68-70 73-74
73-75 73-76 74-78 74-79 75-77 80-81 80-82 80-83 81-85 81-86 82-84
exact/norm bonds :
1-2 1-3 1-4 1-5 5-30 8-12 10-16 13-18 14-19 15-20 16-21 19-22 19-24
21-23 21-25 32-35 34-37 35-41 37-38 37-39 44-48 46-52 50-54 52-55 59-62
61-64 66-69 67-71
67-72 73-76 74-78 74-79 80-83 81-85 81-86
exact bonds :
6-8 6-14 7-10 7-15 8-9 9-13 10-11 11-17 31-32 31-34 32-33 33-36 42-44
42-50 43-46 43-51 44-45 45-49 46-47 47-53 58-59 58-61 59-60 60-63 66-67
66-68 68-70

```
73-74  73-75  75-77  80-81  80-82  82-84


G1:[*1],[*2]

Connectivity :
24:1 X maximum RC ring/chain  25:1 X maximum RC ring/chain
Match level :
1:CLASS  2:CLASS  3:CLASS  4:CLASS  5:CLASS  6:CLASS  7:CLASS  8:CLASS  9:CLASS
10:CLASS  11:CLASS  12:CLASS  13:CLASS  14:CLASS  15:CLASS  16:CLASS  17:CLASS
18:CLASS  19:CLASS
20:CLASS  21:CLASS  22:CLASS  23:CLASS  24:CLASS  25:CLASS  30:CLASS  31:CLASS
32:CLASS  33:CLASS
34:CLASS  35:CLASS  36:CLASS  37:CLASS  38:CLASS  39:CLASS  41:CLASS  42:CLASS
43:CLASS  44:CLASS
45:CLASS  46:CLASS  47:CLASS  48:CLASS  49:CLASS  50:CLASS  51:CLASS  52:CLASS
53:CLASS  54:CLASS
55:CLASS  58:CLASS  59:CLASS  60:CLASS  61:CLASS  62:CLASS  63:CLASS  64:CLASS
66:CLASS  67:CLASS
68:CLASS  69:CLASS  70:CLASS  71:CLASS  72:CLASS  73:CLASS  74:CLASS  75:CLASS
76:CLASS  77:CLASS
78:CLASS  79:CLASS  80:CLASS  81:CLASS  82:CLASS  83:CLASS  84:CLASS  85:CLASS
86:CLASS



L1      STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1              STR

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

Structure attributes must be viewed using STN Express query preparation.


=> s l1
SAMPLE SEARCH INITIATED 10:33:56 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -        0 TO ITERATE

100.0% PROCESSED        0 ITERATIONS                                0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:            0 TO        0
PROJECTED ANSWERS:               0 TO        0

L2            0 SEA SSS SAM L1

=>
Uploading C:\Program Files\STNEXP\Queries\10821739monophosphate2.str
```



chain nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
24 25 30 31 32 33 34 35 36 37 38 39 41 42 43 44 45 46 47 48 49
50 51 52 53
54 55 58 59 60 61 62 63 64 66 67 68 69 70 71 72 73 74 75 76 77
78 79 80
81 82 83 84 85 86
chain bonds :
1-2 1-3 1-4 1-5 5-30 6-8 6-14 7-10 7-15 8-9 8-12 9-13 10-11 10-16
11-17
13-18 14-19 15-20 16-21 19-22 19-24 21-23 21-25 31-32 31-34 32-33 32-35
33-36 34-37
35-41 37-38 37-39 42-44 42-50 43-46 43-51 44-45 44-48 45-49 46-47 46-52
47-53 50-54
52-55 58-59 58-61 59-60 59-62 60-63 61-64 66-67 66-68 66-69 67-71 67-72
68-70 73-74
73-75 73-76 74-78 74-79 75-77 80-81 80-82 80-83 81-85 81-86 82-84
exact/norm bonds :
1-2 1-3 1-4 1-5 5-30 8-12 10-16 13-18 14-19 15-20 16-21 19-22 19-24
21-23 21-25 32-35 34-37 35-41 37-38 37-39 44-48 46-52 50-54 52-55 59-62
61-64 66-69 67-71
67-72 73-76 74-78 74-79 80-83 81-85 81-86
exact bonds :
6-8 6-14 7-10 7-15 8-9 9-13 10-11 11-17 31-32 31-34 32-33 33-36 42-44

```
42-50  43-46  43-51  44-45  45-49  46-47  47-53  58-59  58-61  59-60  60-63  66-67
66-68  68-70
73-74  73-75  75-77  80-81  80-82  82-84


G1:[*1],[*2],[*3],[*4],[*5],[*6],[*7],[*8],[*9]

Connectivity :
24:1 X maximum RC ring/chain  25:1 X maximum RC ring/chain
Match level :
1:CLASS  2:CLASS  3:CLASS  4:CLASS  5:CLASS  6:CLASS  7:CLASS  8:CLASS  9:CLASS
10:CLASS  11:CLASS  12:CLASS  13:CLASS  14:CLASS  15:CLASS  16:CLASS  17:CLASS
18:CLASS  19:CLASS
20:CLASS  21:CLASS  22:CLASS  23:CLASS  24:CLASS  25:CLASS  30:CLASS  31:CLASS
32:CLASS  33:CLASS
34:CLASS  35:CLASS  36:CLASS  37:CLASS  38:CLASS  39:CLASS  41:CLASS  42:CLASS
43:CLASS  44:CLASS
45:CLASS  46:CLASS  47:CLASS  48:CLASS  49:CLASS  50:CLASS  51:CLASS  52:CLASS
53:CLASS  54:CLASS
55:CLASS  58:CLASS  59:CLASS  60:CLASS  61:CLASS  62:CLASS  63:CLASS  64:CLASS
66:CLASS  67:CLASS
68:CLASS  69:CLASS  70:CLASS  71:CLASS  72:CLASS  73:CLASS  74:CLASS  75:CLASS
76:CLASS  77:CLASS
78:CLASS  79:CLASS  80:CLASS  81:CLASS  82:CLASS  83:CLASS  84:CLASS  85:CLASS
86:CLASS



L3      STRUCTURE UPLOADED

=> s l3
SAMPLE SEARCH INITIATED 10:35:02 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -        1 TO ITERATE

100.0% PROCESSED        1 ITERATIONS                                0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:            1 TO       80
PROJECTED ANSWERS:               0 TO        0

L4            0 SEA SSS SAM L3

=>
Uploading C:\Program Files\STNEXP\Queries\10821739monophosphate3.str
```

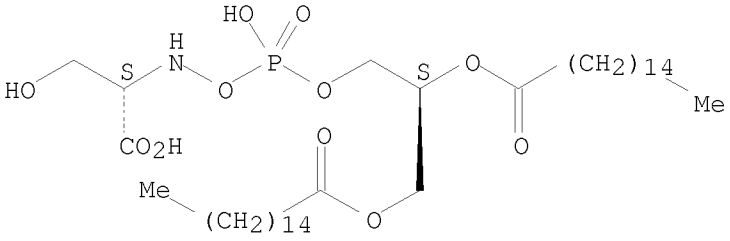


chain nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
24 25 30 31 32 33 34 35 36 37 38 39 41 42 43 44 45 46 47 48 49
50 51 52 53
54 55 58 59 60 61 62 63 64 66 67 68 69 70 71 72 73 74 75 76 77
78 79 80
81 82 83 84 85 86
chain bonds :
1-2 1-3 1-4 1-5 5-30 6-8 6-14 7-10 7-15 8-9 8-12 9-13 10-11 10-16
11-17
13-18 14-19 15-20 16-21 19-22 19-24 21-23 21-25 31-32 31-34 32-33 32-35
33-36 34-37
35-41 37-38 37-39 42-44 42-50 43-46 43-51 44-45 44-48 45-49 46-47 46-52
47-53 50-54
52-55 58-59 58-61 59-60 59-62 60-63 61-64 66-67 66-68 66-69 67-71 67-72
68-70 73-74
73-75 73-76 74-78 74-79 75-77 80-81 80-82 80-83 81-85 81-86 82-84
exact/norm bonds :
1-2 1-3 1-4 1-5 5-30 8-12 10-16 13-18 14-19 15-20 16-21 19-22 19-24
21-23 21-25 32-35 34-37 35-41 37-38 37-39 44-48 46-52 50-54 52-55 59-62
61-64 66-69 67-71
67-72 73-76 74-78 74-79 80-83 81-85 81-86
exact bonds :
6-8 6-14 7-10 7-15 8-9 9-13 10-11 11-17 31-32 31-34 32-33 33-36 42-44

```
42-50  43-46  43-51  44-45  45-49  46-47  47-53  58-59  58-61  59-60  60-63  66-67
66-68  68-70
73-74  73-75  75-77  80-81  80-82  82-84


G1:[*1],[*2],[*3],[*4],[*5],[*6],[*7],[*8],[*9]

Connectivity :
24:1 X maximum RC ring/chain  25:1 X maximum RC ring/chain  54:1 X maximum RC
ring/chain
55:1 X maximum RC ring/chain  64:1 X maximum RC ring/chain
Match level :
1:CLASS  2:CLASS  3:CLASS  4:CLASS  5:CLASS  6:CLASS  7:CLASS  8:CLASS  9:CLASS
10:CLASS  11:CLASS  12:CLASS  13:CLASS  14:CLASS  15:CLASS  16:CLASS  17:CLASS
18:CLASS  19:CLASS
20:CLASS  21:CLASS  22:CLASS  23:CLASS  24:CLASS  25:CLASS  30:CLASS  31:CLASS
32:CLASS  33:CLASS
34:CLASS  35:CLASS  36:CLASS  37:CLASS  38:CLASS  39:CLASS  41:CLASS  42:CLASS
43:CLASS  44:CLASS
45:CLASS  46:CLASS  47:CLASS  48:CLASS  49:CLASS  50:CLASS  51:CLASS  52:CLASS
53:CLASS  54:CLASS
55:CLASS  58:CLASS  59:CLASS  60:CLASS  61:CLASS  62:CLASS  63:CLASS  64:CLASS
66:CLASS  67:CLASS
68:CLASS  69:CLASS  70:CLASS  71:CLASS  72:CLASS  73:CLASS  74:CLASS  75:CLASS
76:CLASS  77:CLASS
78:CLASS  79:CLASS  80:CLASS  81:CLASS  82:CLASS  83:CLASS  84:CLASS  85:CLASS
86:CLASS



L5      STRUCTURE UPLOADED

=> s l5
SAMPLE SEARCH INITIATED 10:36:24 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -        1 TO ITERATE

100.0% PROCESSED        1 ITERATIONS                                0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:            1 TO       80
PROJECTED ANSWERS:               0 TO        0

L6            0 SEA SSS SAM L5

=> s l5 sss full
FULL SEARCH INITIATED 10:36:30 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -       23 TO ITERATE

100.0% PROCESSED       23 ITERATIONS                                2 ANSWERS
SEARCH TIME: 00.00.01

L7            2 SEA SSS FUL L5

=> d l7 scan

L7   2 ANSWERS   REGISTRY  COPYRIGHT 2009 ACS on STN
IN   Hexadecanoic acid, (1S)-1-[[[[[[(1S)-1-carboxy-2-
     hydroxyethyl]amino]oxy]hydroxyphosphinyl]oxy]methyl]-1,2-ethanediyl ester
```

(9CI)
MF C38 H74 N O11 P

Absolute stereochemistry.

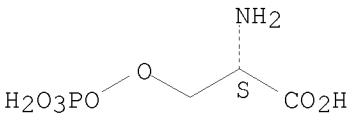


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L7 2 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN
IN L-Alanine, 3-(phosphonodioxy)-
MF C3 H8 N O7 P

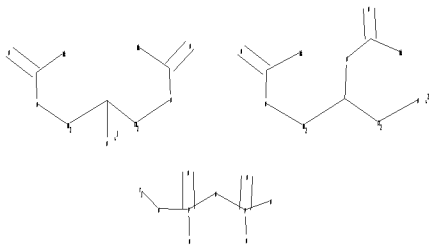
Absolute stereochemistry.

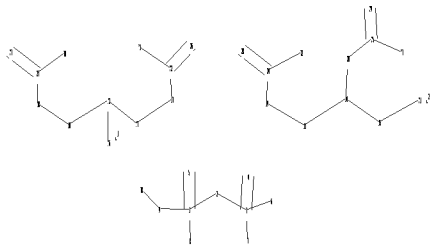


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

ALL ANSWERS HAVE BEEN SCANNED

=>
Uploading C:\Program Files\STNEXP\Queries\10821739pyrophosphate.str





chain nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
24 25 26 27 28 29 30 31 32 33 38
chain bonds :
1-2 1-5 1-8 1-9 2-3 3-4 3-6 3-7 9-38 10-12 10-18 11-14 11-19 12-13
12-16 13-17 14-15 14-20 15-21 17-22 18-23 19-24 20-25 22-26 22-31 23-27
23-30 24-28 24-32
25-29 25-33
exact/norm bonds :
1-2 1-5 1-8 1-9 2-3 3-4 3-6 3-7 9-38 12-16 14-20 17-22 18-23 19-24
20-25 22-26 22-31 23-27 23-30 24-28 24-32 25-29 25-33
exact bonds :
10-12 10-18 11-14 11-19 12-13 13-17 14-15 15-21

G1:[*1],[*2]

Connectivity :
30:1 X maximum RC ring/chain 31:1 X maximum RC ring/chain 32:1 X maximum RC
ring/chain
33:1 X maximum RC ring/chain
Match level :
1:CLASS 2:CLASS 3:CLASS 4:CLASS 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:CLASS
10:CLASS 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 16:CLASS 17:CLASS
18:CLASS 19:CLASS
20:CLASS 21:CLASS 22:CLASS 23:CLASS 24:CLASS 25:CLASS 26:CLASS 27:CLASS
28:CLASS 29:CLASS

30:CLASS 31:CLASS 32:CLASS 33:CLASS 38:CLASS

L8   STRUCTURE UPLOADED

=> s l8
SAMPLE SEARCH INITIATED 10:37:38 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -   0 TO ITERATE

100.0% PROCESSED   0 ITERATIONS   0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS: 0 TO   0
PROJECTED ANSWERS: 0 TO   0

L9   0 SEA SSS SAM L8

=> d l8
L8 HAS NO ANSWERS
L8   STR

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

Structure attributes must be viewed using STN Express query preparation.

=> s l8 sss full
FULL SEARCH INITIATED 10:38:03 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -   1 TO ITERATE

100.0% PROCESSED   1 ITERATIONS   0 ANSWERS
SEARCH TIME: 00.00.01

L10   0 SEA SSS FUL L8

=> file stnguide
COST IN U.S. DOLLARS   SINCE FILE   TOTAL
                                                     ENTRY   SESSION
FULL ESTIMATED COST   374.64   374.86

FILE 'STNGUIDE' ENTERED AT 10:38:12 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 25, 2009 (20090925/UP).

=> file hcaplus
COST IN U.S. DOLLARS   SINCE FILE   TOTAL
                                                     ENTRY   SESSION
FULL ESTIMATED COST   0.63   375.49

FILE 'HCAPLUS' ENTERED AT 10:43:33 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1907 - 1 Oct 2009 VOL 151 ISS 14
FILE LAST UPDATED: 30 Sep 2009 (20090930/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2009

HCAplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2009.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

The ALL, BIB, MAX, and STD display formats in the CA/CAplus family
of databases have been updated to include new citing references
information. This enhancement may impact record import into
database management software. For additional information, refer
to NEWS 9.

```
=> s (lysophosphtidic acid)(4a)(inhib? or antag?)

             0 LYSOPHOSPHTIDIC
       4911805 ACID
             0 LYSOPHOSPHTIDIC ACID
                 (LYSOPHOSPHTIDIC(W)ACID)
       2188443 INHIB?
        334665 ANTAG?
L11          0 (LYSOPHOSPHTIDIC ACID)(4A)(INHIB? OR ANTAG?)

=> s phosphate or phrophosphate or phosphoric

        637370 PHOSPHATE
             4 PHROPHOSPHATE
        121867 PHOSPHORIC
L12     717126 PHOSPHATE OR PHROPHOSPHATE OR PHOSPHORIC

=> s cholesterol or hypercholesterol? or hyperlipid? or atehrosclerosis or neointima

        203298 CHOLESTEROL
         20115 HYPERCHOLESTEROL?
         18730 HYPERLIPID?
             0 ATEHROSCLEROSIS
          2232 NEOINTIMA
L13     220274 CHOLESTEROL OR HYPERCHOLESTEROL? OR HYPERLIPID? OR ATEHROSCLEROS
               IS OR NEOINTIMA

=> s l11 and l12
```

```
L14          0 L11 AND L12

=> s l11 and l13

L15          0 L11 AND L13

=> s l12 and l13

L16       6976 L12 AND L13

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       2.85     378.34

FILE 'STNGUIDE' ENTERED AT 10:43:40 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 25, 2009 (20090925/UP).

=> file hcaplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.07     378.41

FILE 'HCAPLUS' ENTERED AT 10:44:18 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)


Copyright of the articles to which records in this database refer is
held by the publishers listed in the PUBLISHER (PB) field (available
for records published or updated in Chemical Abstracts after December
26, 1996), unless otherwise indicated in the original publications.
The CA Lexicon is the copyrighted intellectual property of the
American Chemical Society and is provided to assist you in searching
databases on STN.  Any dissemination, distribution, copying, or storing
of this information, without the prior written consent of CAS, is
strictly prohibited.

FILE COVERS 1907 - 1 Oct 2009  VOL 151 ISS 14
FILE LAST UPDATED: 30 Sep 2009  (20090930/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Aug 2009

HCAplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate
substance identification.

The ALL, BIB, MAX, and STD display formats in the CA/CAplus family
of databases have been updated to include new citing references
```

information. This enhancement may impact record import into
database management software. For additional information, refer
to NEWS 9.

=> s (lysophosphatidic acid)(4a)(inhib? or antag?)

          3893 LYSOPHOSPHATIDIC
       4911805 ACID
          3121 LYSOPHOSPHATIDIC ACID
                 (LYSOPHOSPHATIDIC(W)ACID)
       2188443 INHIB?
        334665 ANTAG?
L17   314 (LYSOPHOSPHATIDIC ACID)(4A)(INHIB? OR ANTAG?)

=> s phosphate or pyrophosphate or phosphoric

        637370 PHOSPHATE
         44113 PYROPHOSPHATE
        121867 PHOSPHORIC
L18   744163 PHOSPHATE OR PYROPHOSPHATE OR PHOSPHORIC

=> s cholesterol or hypercholesterol? or hyperlipid? or atehrosclerosis or neointima

        203298 CHOLESTEROL
         20115 HYPERCHOLESTEROL?
         18730 HYPERLIPID?
             0 ATEHROSCLEROSIS
          2232 NEOINTIMA
L19   220274 CHOLESTEROL OR HYPERCHOLESTEROL? OR HYPERLIPID? OR ATEHROSCLEROS
               IS OR NEOINTIMA

=> s l17 and l18

L20   62 L17 AND L18

=> s l17 and l19

L21   3 L17 AND L19

=> s l18 and l19

L22   7389 L18 AND L19

=> file stnguide

COST IN U.S. DOLLARS   SINCE FILE   TOTAL
                                                     ENTRY   SESSION
FULL ESTIMATED COST   2.85   381.26

FILE 'STNGUIDE' ENTERED AT 10:44:25 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 25, 2009 (20090925/UP).

=> d l21 1-3 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L21 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Anti-atherosclerotic molecules targeting oxidative stress and inflammation
AB A review. The accumulation of lipids within arteries remains to be the initial impulse for the pathogenesis of atherosclerosis; however, both inflammation and oxidative stress are considered to play a critical role in this process. Several lipid lowering drugs are used as the first line therapy in atherosclerosis; however, different agents have been found to exhibit beneficial effects which are independent of their lipid lowering activity. Both statins and fibrates have been reported to exert anti-inflammatory and anti-oxidative effects in addition to their anti-atherosclerotic actions. Furthermore, anti-hypertensive, anti-diabetic and anti-platelet drugs, which reduce oxidative stress and inflammation, have been shown to attenuate atherosclerosis. In addition, novel substances such as HDL-related agents, cyclopentenone prostaglandins, lipoprotein-associated phospholipase A2 inhibitors, 5-lipoxygenase pathway inhibitors, acyl CoA: cholesterol acyltransferase inhibitors, analogs of probucol and lysophosphatidic acid antagonists have been developed for the treatment of atherosclerosis as a consequence of their actions on oxidative stress and inflammation. The present article reviews the involvement of inflammation and oxidative stress in the pathogenesis of atherosclerosis and focuses on the mechanisms of some clin. used as well as potential anti-atherosclerotic substances with anti-inflammatory and anti-oxidative properties.
AN 2009:1022565 HCAPLUS <<LOGINID::20091001>>
DN 151:235384
TI Anti-atherosclerotic molecules targeting oxidative stress and inflammation
AU Adameova, A.; Xu, Y. J.; Duhamel, T. A.; Tappia, P. S.; Shan, L.; Dhalla, N. S.
CS Institute of Cardiovascular Sciences, St. Boniface General Hospital Research Centre, Faculty of Medicine, University of Manitoba, Winnipeg, Can.
SO Current Pharmaceutical Design (2009), 15(27), 3094-3107
CODEN: CPDEFP; ISSN: 1381-6128
PB Bentham Science Publishers Ltd.
DT Journal; General Review
LA English
RE.CNT 159 THERE ARE 159 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Treatment of viral infections by modulation of host cell metabolic pathways
AB Alterations of certain metabolite concns. and fluxes that occur in response to viral infection are described. Host cell enzymes in the involved metabolic pathways are selected as targets for intervention; i.e., to restore metabolic flux to disadvantage viral replication, or to further derange metabolic flux resulting in "suicide" of viral-infected cells (but not uninfected cells) in order to limit viral propagation. While any of the enzymes in the relevant metabolic pathway can be selected, pivotal enzymes at key control points in these metabolic pathways are preferred as candidate antiviral drug targets. Inhibitors of these enzymes are used to reverse, or redirect, the effects of the viral infection. Drug candidates are tested for antiviral activity using screening assays in vitro and host cells, as well as in animal models. Animal models are then used to test efficacy of candidate compds. in preventing and treating viral infections. The antiviral activity of enzyme inhibitors is demonstrated.
AN 2009:198413 HCAPLUS <<LOGINID::20091001>>
DN 150:252581

TI Treatment of viral infections by modulation of host cell metabolic pathways
IN Shenk, Thomas; Rabinowitz, Joshua D.; Munger, Josh; Bennett, Bryson
PA The Trustees of Princeton University, USA
SO PCT Int. Appl., 339pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2009023059	A2	20090219	WO 2008-US6959	20080602
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20090239830	A1	20090924	US 2008-156517	20080602
PRAI US 2007-932769P	P	20070601		
US 2008-33243P	P	20080303		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 150:252581

L21 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPARγ that inhibit PPARγ signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.
AN 2004:857161 HCAPLUS <<LOGINID::20091001>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

```
PI   US 20040204383       A1    20041014    US 2004-821739         20040409
     AU 2004229467        A1    20041028    AU 2004-229467         20040409
     AU 2004229467        B2    20070125
     CA 2521189           A1    20041028    CA 2004-2521189        20040409
     WO 2004091496        A2    20041028    WO 2004-US11016        20040409
     WO 2004091496        A3    20050324
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
             SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
             TD, TG
     EP 1613298           A2    20060111    EP 2004-759365         20040409
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
     JP 2007525449        T     20070906    JP 2006-509874         20040409
PRAI US 2003-462274P      P     20030411
     WO 2004-US11016      W     20040409


=> file hcaplus
COST IN U.S. DOLLARS                              SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.07     393.25

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)        SINCE FILE      TOTAL
                                                     ENTRY    SESSION
CA SUBSCRIBER PRICE                                   0.00      -2.46

FILE 'HCAPLUS' ENTERED AT 10:45:32 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)
```


FILE COVERS 1907 - 1 Oct 2009 VOL 151 ISS 14
FILE LAST UPDATED: 30 Sep 2009 (20090930/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2009

HCAplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate substance identification.

The ALL, BIB, MAX, and STD display formats in the CA/CAplus family of databases have been updated to include new citing references information. This enhancement may impact record import into database management software. For additional information, refer to NEWS 9.

```
=> s l20 and (PY<2003 or AY<2003 or PRY<2003)
      22985329 PY<2003
       4511505 AY<2003
       3981271 PRY<2003
L23         32 L20 AND (PY<2003 OR AY<2003 OR PRY<2003)

=> d l23 1-32 ti abs bib hitstr
```

L23 ANSWER 1 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN

TI The phospholipids sphingosine-1-phosphate and lysophosphatidic acid prevent apoptosis in osteoblastic cells via a signaling pathway involving Gi proteins and phosphatidylinositol-3 kinase

AB The naturally occurring phospholipids lysophosphatidic acid (LPA) and sphingosine-1-phosphate (S1P) have recently emerged as bioactive compds. that exert mitogenic effects in many cell types, including osteoblasts. In the current study, we examined the ability of each of these compds. to influence osteoblast survival. Using terminal deoxynucleotidyl transferase-mediated deoxyuridine 5'-triphosphate nick-end labeling and DNA fragmentation assays, we found that both LPA and S1P dose-dependently inhibited (by at least 50% and 40%, resp.) the apoptosis induced by serum withdrawal in cultures of primary calvarial rat osteoblasts and SaOS-2 cells. The antiapoptotic effects were inhibited by pertussis toxin, wortmannin, and LY294002, implicating Gi proteins and phosphatidylinositol-3 kinase (PI-3 kinase) in the signaling pathway that mediates phospholipid-induced osteoblast survival. Specific inhibitors of p42/44 MAPK signaling did not block LPA- or S1P-induced osteoblast survival. LPA and S1P induced PI-3 kinase-dependent activation of p70 S6 kinase, but rapamycin, a specific inhibitor of p70 S6 kinase activation, did not prevent phospholipid-induced osteoblast survival. LPA and S1P also inhibited apoptosis in Swiss 3T3 fibroblastic cells in a Gi protein-dependent fashion. In fibroblastic cells, however, the antiapoptotic effects of S1P were sensitive to inhibition of both PI-3 kinase and p42/44 MAPK signaling, whereas those of LPA were partially abrogated by inhibitors of p42/44 MAPK signaling but not by PI-3 kinase inhibitors. These data demonstrate that LPA and S1P potently promote osteoblast survival in vitro, and that cell-type specificity exists in the antiapoptotic signaling pathways activated by phospholipids.

AN 2002:923658 HCAPLUS <<LOGINID::20091001>>

DN 138:318116

TI The phospholipids sphingosine-1-phosphate and lysophosphatidic acid prevent apoptosis in osteoblastic cells via a signaling pathway involving Gi proteins and phosphatidylinositol-3 kinase

AU Grey, Andrew; Chen, Qi; Callon, Karen; Xu, Xin; Reid, Ian R.; Cornish, Jill

CS Department of Medicine, University of Auckland, Auckland, N. Z.

SO Endocrinology (2002), 143(12), 4755-4763
CODEN: ENDOAO; ISSN: 0013-7227

PB Endocrine Society

DT Journal

LA English

OSC.G 37 THERE ARE 37 CAPLUS RECORDS THAT CITE THIS RECORD (37 CITINGS)

RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 2 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Comparative analysis of human and rat S1P5 (edg8): differential expression profiles and sensitivities to antagonists
AB Five guanine nucleotide-binding protein-coupled receptors (S1P1-5) for the lysophospholipid mediator sphingosine 1-phosphate (S1P) have thus far been described. Whereas tissue distribution and functional properties of the human S1P1-4 genes are well characterized, only limited functional and expression data are available for S1P5, to date. Northern blot anal. indicated that human S1P5 (hS1P5) is an alternatively spliced gene, with a 5.4-kb transcript that is predominantly expressed in peripheral tissues, and a 2.4-kb transcript expressed in brain, spleen, and peripheral blood leukocytes. In contrast, rat S1P5 (rS1P5) was exclusively detected in brain and skin. Expression of hS1P5 and rS1P5 in mammalian CHO-K1 or HEK293 cells conferred onto the cells the ability to mobilize intracellular calcium as determined by a functional Fluorometric Imaging Plate Reader assay, when challenged with S1P and dihydro S1P, resp. Applying a lipid library with 200 bioactive lipids in a functional Fluorometric Imaging Plate Reader assay did not reveal addnl. agonists. However, both receptors exhibited differential sensitivity towards the S1P- and lysophosphatidic acid-receptor antagonist, suramin: rS1P5-mediated intracellular calcium mobilization was partly inhibited by suramin (IC50: 5800 μM), whereas hS1P5 was completely antagonized (IC50: 130 μM). Both receptors were sensitive towards inhibition with the related drug (8,8'-(carbonylbis(imino-3,1-phenylene))bis(1,3,5-naphthalenetrisulfonicacid))but ic50 values differed significantly (340 μM for hS1P5, 4000 μM for rS1P5). In addition, rS1P5 displayed antiproliferative effects in transfected CHO-K1 and HEK293 cells in contrast to hS1P5. Taken together, our data imply that differences between hS1P5 and rS1P5 will be an important point to be considered in the development of selective receptor antagonists.
AN 2002:701945 HCAPLUS <<LOGINID::20091001>>
DN 138:317912
TI Comparative analysis of human and rat S1P5 (edg8): differential expression profiles and sensitivities to antagonists
AU Niedernberg, Anke; Scherer, Constanze R.; Busch, Andreas E.; Kostenis, Evi
CS Disease group Cardiovascular, Frankfurt, 65926, Germany
SO Biochemical Pharmacology (2002), 64(8), 1243-1250
CODEN: BCPCA6; ISSN: 0006-2952
PB Elsevier Science Inc.
DT Journal
LA English
OSC.G 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS RECORD (10 CITINGS)
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 3 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Modification of the antitumor effect of doxorubicin by phosphorylated retinoids conjugated to α-fetoprotein
AB The influence of new retinoids representing all-trans-retinoic acid (RA) amides with O-phosphates of ethanolamine, L-serine, L-threonine (Thr), and L-tyrosine conjugated to α-fetoprotein on the antitumor action of the antibiotic doxorubicin was evaluated with regard to inoculated Ehrlich ascitic carcinoma. The synthesized compds. are structurally analogous to N-palmitoyl-O-phospho-L-serine (NP-Ser-PA) and N-palmitoyl-O-phospho-L-tyrosine (NP-Tyr-PA), which are antagonists of the receptors of lysophosphatidic acid. The complex of doxorubicin with NR-Thr-PA in the presence

of α-FP displayed a lower antitumor activity compared to that of free doxorubicin, in which the Ehrlich carcinoma cell growth was inhibited by 37%. The RA complex containing only NR-Tyr-PA produced a more evident cytotoxic action upon the tumor compared to both the complex and free doxorubicin. An optimum composition was offered by the doxorubicin complex with NR-Tyr-PA and α-FP produced a pos. influence on the antitumor properties of this complex. The absence of a reliable effect for the mixture of doxorubicin with NR-Tyr-PA is attributed by the formation of an ion complex between the components, related to the interaction between the amino group of daunosamine and the phosphate moiety of NR-Tyr-PA.

AN 2002:628766 HCAPLUS <<LOGINID::20091001>>
DN 138:147338
TI Modification of the antitumor effect of doxorubicin by phosphorylated retinoids conjugated to α-fetoprotein
AU Arsenov, D. V.; Babitskaya, S. V.; Vashkevich, I. I.; Dad'kov, I. D.; Kisel', M. A.; Kuz'mitskii, B. B.; Strel'chenok, O. A.
CS Institute of Bioorganic Chemistry, National Academy of Sciences of Belarus, Minsk, Belarus
SO Pharmaceutical Chemistry Journal (Translation of Khimiko-Farmatsevticheskii Zhurnal) (2001), 35(12), 657-660
CODEN: PCJOAU; ISSN: 0091-150X
PB Kluwer Academic/Consultants Bureau
DT Journal
LA English
RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 4 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Molecular modeling of lysophosphatidic acid receptor antagonists
AB Lysophosphatidic acid (LPA) elicits a variety of responses including mitogenesis, cytoskeletal changes, activation of Ca2+ transients, and effects on apoptosis. These responses are elicited via LPA1/EDG2, LPA2/EDG4, and LPA3/EDG7 G protein-coupled receptors. These receptors are members of the endothelial differentiation gene family. In order to understand the physiol. significance of LPA highly selective antagonists are necessary. Recently, dioctyl glycerol pyrophosphate (DGPP) and fatty alkyl phosphate (FAP) were shown to be potent and selective antagonists towards LPA3 receptor. We have docked DGPP and FAP in our LPA1, LPA2, and LPA3 receptor models and our docked energies agree with the observed trend in inhibition consts. (Ki). The docked positions of the antagonist relative to the agonist overlap in the position of the polar head group, but diverge in the favored position of the hydrophobic tail(s).
AN 2002:617933 HCAPLUS <<LOGINID::20091001>>
TI Molecular modeling of lysophosphatidic acid receptor antagonists
AU Sardar, Vineet M.; Virag, Tamas; Fischer, David J.; Elrod, Don; Bautista, Debra L.; Wang, De-an; Nusser, Nora; Yokoyama, Kazuaki; Baker, Daniel L.; Miller, Duane D.; Tigyi, Gabor; Parrill, Abby L.
CS Department of Chemistry and Computational Research on Materials Institute, University of Memphis, Memphis, TN, 38152-6060, USA
SO Abstracts of Papers, 224th ACS National Meeting, Boston, MA, United States, August 18-22, 2002 (2002), MEDI-079 Publisher: American Chemical Society, Washington, D. C.
CODEN: 69CZPZ
DT Conference; Meeting Abstract
LA English

L23 ANSWER 5 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN

TI Lysophosphatidic acid inhibition of the accumulation of Pseudomonas aeruginosa PAO1 alginate, pyoverdin, elastase and LasA

AB The pathogenesis of Pseudomonas aeruginosa is at least partially attributable to its ability to synthesize and secrete the siderophore pyoverdin and the two zinc metalloproteases elastase and LasA, and its ability to form biofilms in which bacterial cells are embedded in an alginate matrix. In the present study, a lysophospholipid, 1-palmitoyl-2-hydroxy-sn-glycero-3-phosphate [also called monopalmitoylphosphatidic acid (MPPA)], which accumulates in inflammatory exudates, was shown to inhibit the extracellular accumulation of P. aeruginosa PAO1 alginate, elastase, LasA protease and the siderophore pyoverdin. MPPA also inhibited biofilm formation. The inhibitory effects of MPPA occur independently of rpoS expression and without affecting the accumulation of the autoinducers N-(3-oxododecanoyl) homoserine lactone and N-butyryl-L-homoserine lactone, and may be due, at least in part, to the ability of MPPA to bind divalent cations.

AN 2002:484327 HCAPLUS <<LOGINID::20091001>>

DN 138:86340

TI Lysophosphatidic acid inhibition of the accumulation of Pseudomonas aeruginosa PAO1 alginate, pyoverdin, elastase and LasA

AU Laux, David C.; Corson, Joy M.; Givskov, Michael; Hentzer, Morten; Moller, Annette; Wosencroft, Kathleen A.; Olson, Joan C.; Krogfelt, Karen A.; Goldberg, Joanna B.; Cohen, Paul S.

CS Department of Cell and Molecular Biology, University of Rhode Island, Kingston, RI, 02881, USA

SO Microbiology (Reading, United Kingdom) (2002), 148(6), 1709-1723 CODEN: MROBEO; ISSN: 1350-0872

PB Society for General Microbiology

DT Journal

LA English

OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

RE.CNT 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 6 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN

TI Molecular basis for lysophosphatidic acid receptor antagonist selectivity

AB A review. Recent characterization of lysophosphatidic acid (LPA) receptors has made possible studies elucidating the structure-activity relationships (SAR) for agonist activity at individual receptors. Addnl., the availability of these receptors has allowed the identification of antagonists of LPA-induced effects. Two receptor-subtype selective LPA receptor antagonists, one selective for the LPA1/EDG2 receptor (a benzyl-4-oxybenzyl N-acyl ethanolamide phosphate, NAEPA, derivative) and the other selective for the LPA3/EDG7 receptor (diacylglycerol pyrophosphate, DGPP, 8:0), have recently been reported. The receptor SAR for both agonists and antagonists are reviewed, and the mol. basis for the difference between agonism and antagonism as well as for receptor-subtype antagonist selectivity identified by mol. modeling is described. The implications of the newly available receptor-subtype selective antagonists are also discussed.

AN 2002:459276 HCAPLUS <<LOGINID::20091001>>

DN 138:86877

TI Molecular basis for lysophosphatidic acid receptor antagonist selectivity

AU Sardar, Vineet M.; Bautista, Debra L.; Fischer, David J.; Yokoyama, Kazuaki; Nusser, Nora; Virag, Tamas; Wang, De-an; Baker, Daniel L.; Tigyi, Gabor; Parrill, Abby L.

CS Department of Chemistry and Computational Research on Materials Institute,

The University of Memphis, Memphis, TN, 38152-6060, USA
SO Biochimica et Biophysica Acta, Molecular and Cell Biology of Lipids (2002), 1582(1-3), 309-317
CODEN: BBMLFG; ISSN: 1388-1981
PB Elsevier B.V.
DT Journal; General Review
LA English
OSC.G 52 THERE ARE 52 CAPLUS RECORDS THAT CITE THIS RECORD (53 CITINGS)
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 7 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Albumin stimulates lysophosphatidic acid acyltransferase activity in T-lymphocyte membranes
AB Phosphatidic acid (PtdOH) and lysophosphatidic acid (lysoPtdOH) have been shown to enhance T-lymphocyte function. However, the FA preference and influence of acyl-CoA binding proteins on lysoPtdOH and PtdOH biosynthesis are not known. Therefore, we determined glycerol-3-phosphate acyltransferase (GPAT) and lysophosphatidic acid acyltransferase (LAT) activity in rat T-lymphocyte and liver membrane prepns. in the presence of palmitoyl-CoA and oleoyl-CoA with or without BSA. We found two different properties of GPAT and LAT in whole T-lymphocyte membrane prepns. relative to liver. First, T-lymphocyte basal GPAT and LAT activities were similar, whereas in liver membranes LAT activity was 10-fold higher than GPAT. Second, T-lymphocyte LAT, but not GPAT, activity was inducible (fivefold) by the addition of albumin in the presence of palmitoyl-CoA but not oleoyl-CoA. In contrast, albumin stimulated GPAT, but not LAT, activity in liver membranes in the presence of palmitoyl-CoA. These results show, for the first time, that T-lymphocyte LAT activity can be increased by the presence of an acyl-CoA binding protein, which may indicate a new important control mechanism for regulating intracellular lysoPtdOH and PtdOH levels in T-lymphocytes.
AN 2002:396235 HCAPLUS <<LOGINID::20091001>>
DN 137:198675
TI Albumin stimulates lysophosphatidic acid acyltransferase activity in T-lymphocyte membranes
AU Jolly, Christopher A.; Kannan, Latha
CS Division of Nutritional Sciences and Institute for Cellular and Molecular Biology, The University of Texas at Austin, Austin, TX, 78712, USA
SO Lipids (2002), 37(5), 475-480
CODEN: LPDSAP; ISSN: 0024-4201
PB AOCS Press
DT Journal
LA English
OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 8 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Noradrenaline release-inhibiting receptors on PC12 cells devoid of α2- and CB1 receptors: similarities to presynaptic imidazoline and edg receptors
AB The aim of the present study was to classify release-inhibiting receptors on rat pheochromocytoma PC12 cells. Veratridine-evoked [3H]noradrenaline release from PC12 cells was inhibited by micromolar concns. of the imidazoline and guanidine derivs. cirazoline, clonidine, aganodine, 1,3-di(2-tolyl)guanidine, BDF6143 and agmatine, and of the cannabinoid receptor agonist WIN55,212-2 (R(+)-[2,3-dihydro-5-methyl-3-[(morpholinyl)methyl]pyrrolo-[1,2,3-de]-1,4-benzoxazin-yl](1-naphthalenyl)methanone mesylate), but not by noradrenaline. The inhibitory effect of clonidine was antagonized by micromolar concns. of

rauwolscine and SR141716A (N-[piperidin-1-yl]-5-[4-chlorophenyl]-1-[2,4-dichlorophenyl]-4-methyl-1H-pyrazole-3-carboxamide). The potencies of the agonists and antagonists were compatible with an action at previously characterized presynaptic imidazoline receptors. 1-Oleoyl-lysophosphatidic acid, but not sphingosine-1-phosphate, produced an inhibition of release that was antagonized by 30 μM rauwolscine, 1 μM SR141716A and 10 μM LY320135 as well as by pretreatment of the cells with 100 μM clonidine for 72 h. Polymerase chain reaction (PCR) expts. on cDNA from PC12 mRNA suggest mRNA expression of lysophospholipid receptors encoded by the genes edg2, edg3, edg5 and edg7, but not of receptors encoded by edg1, edg4, edg6 and edg8, and not of α2A-nd CB1 receptors. In conclusion, PC12 cells are not endowed with α2-adrenoceptors and CB1 cannabinoid receptors, but with an inhibitory receptor recognizing imidazolines, guanidines and WIN55,212-2 similar to that on sympathetic nerves. The PCR results and the ability of 1-oleoyl-LPA to mimic these drugs (also with respect to their susceptibility to antagonists) suggest that the release-inhibiting receptor may be an edg-encoded lysophospholipid receptor.

AN 2001:883810 HCAPLUS <<LOGINID::20091001>>
DN 136:319263
TI Noradrenaline release-inhibiting receptors on PC12 cells devoid of α2- and CB1 receptors: similarities to presynaptic imidazoline and edg receptors
AU Molderings, G. J.; Bonisch, H.; Hammermann, R.; Gothert, M.; Bruss, M.
CS Institute of Pharmacology and Toxicology, University of Bonn, Bonn, 53113, Germany
SO Neurochemistry International (2002), 40(2), 157-167
CODEN: NEUIDS; ISSN: 0197-0186
PB Elsevier Science Ltd.
DT Journal
LA English
OSC.G 21 THERE ARE 21 CAPLUS RECORDS THAT CITE THIS RECORD (21 CITINGS)
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 9 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Short-chain phosphatidates are subtype-selective antagonists of lysophosphatidic acid receptors
AB Lysophosphatidic acid (LPA) and sphingosine-1-phosphate (S1P) are members of the phospholipid growth factor family. A major limitation in the field to date has been a lack of receptor subtype-specific agonists and antagonists. Here, we report that dioctylglycerol pyrophosphate and dioctylphosphatidic acid are selective antagonists of the LPA1 and LPA3 receptors, but prefer LPA3 by an order of magnitude. Neither mol. had an agonistic or antagonistic effect on LPA2 receptor. Consistent with this receptor subtype selectivity, dioctylglycerol pyrophosphate inhibited cellular responses to LPA in NiH3T3 fibroblasts, HEY ovarian cancer cells, PC12 pheochromocytoma cells, and Xenopus laevis oocytes. Responses elicited by S1P in these cell lines that endogenously express S1P1, S1P2, S1P3, and S1P5 receptors were unaffected by dioctylglycerol pyrophosphate. Responses evoked by the G protein-coupled receptor ligands acetylcholine, serotonin, ATP, and thrombin receptor-activating peptide were similarly unaffected, suggesting that the short-chain phosphatidates are receptor subtype-specific lysophosphatidate antagonists.
AN 2001:723801 HCAPLUS <<LOGINID::20091001>>
DN 136:96559
TI Short-chain phosphatidates are subtype-selective antagonists of lysophosphatidic acid receptors
AU Fischer, David J.; Nusser, Nora; Virag, Tamas; Yokoyama, Kazuaki; Wang, De-An; Baker, Daniel L.; Bautista, Debra; Parrill, Abby L.; Tigyi, Gabor

CS Department of Physiology, University of Tennessee Health Science Center, Memphis, TN, USA
SO Molecular Pharmacology (2001), 60(4), 776-784
CODEN: MOPMA3; ISSN: 0026-895X
PB American Society for Pharmacology and Experimental Therapeutics
DT Journal
LA English
OSC.G 89 THERE ARE 89 CAPLUS RECORDS THAT CITE THIS RECORD (89 CITINGS)
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 10 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation
AB The present invention relates to lysophosphatidic acid (LPA) analogs and cyclic derivs. of the analogs as well as pharmaceutical compns. which include those compds. Also disclosed are methods of using such compds., which have activity as agonists or as antagonists of LPA receptors; such methods including inhibiting LPA activity on an LPA receptor, modulating LPA receptor activity, treating cancer, enhancing cell proliferation, and treating a wound. Thus, 2-amino-3-oxo-3-(tetradecylamino)propyl dihydrogen phosphate (I), 2-(acetylamino)-3-oxo-3-(tetradecylamino)propyl dihydrogen phosphate (II), and 1,2-(3-octadecyloxypropane)-bis(dihydrogen phosphate) (III) were synthesized. The cytotoxicity of these compds. on prostate cancer cell lines was determined The IC50's observed were 0.7
± 0.1 for I on PC-3 cells, 0.7 ± 0.1 for II on DU145 cells, and 3.1 ± 3.2 for III on LNCaP cells. Addnl., phosphoric acid monododecyl ester (IV) was prepared and screened in Xenopus oocytes (which produce the PSP24 receptor) and in recombinant RH7777 cells producing Edg-2, Edg-4, and Edg-7 receptors. In Xenopus IV inhibited LPA-induced chloride currents with an IC50 value of about 8.1 nM. In Edg-2 and Edg-4-expressing RH7777 cells IV significantly inhibited the Ca2+ responses while in Edg-7-expressing cells this compound increased the Ca2+ responses.
AN 2001:713600 HCAPLUS <<LOGINID::20091001>>
DN 135:267219
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation
IN Miller, Duane D.; Tigyi, Gabor; Dalton, James T.; Sardar, Vineet M.; Elrod, Don B.; Xu, Huiping; Baker, Daniel L.; Wang, Dean; Liliom, Karoly; Fischer, David J.; Virag, Tamas; Nusser, Nora
PA University of Tennessee Research Corporation, USA
SO PCT Int. Appl., 140 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001071022	A2	20010927	WO 2001-US8729	20010319 <--
WO 2001071022	A3	20020404		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2402038 A1 20010927 CA 2001-2402038 20010319 <--
AU 2001049263 A 20011003 AU 2001-49263 20010319 <--
EP 1263752 A2 20021211 EP 2001-922465 20010319 <--
EP 1263752 B1 20071205
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2004506604 T 20040304 JP 2001-569403 20010319 <--
AT 380187 T 20071215 AT 2001-922465 20010319 <--
EP 1918287 A2 20080507 EP 2007-122187 20010319 <--
EP 1918287 A3 20080820
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR
ES 2298227 T3 20080516 ES 2001-922465 20010319 <--
KR 874392 B1 20081217 KR 2002-712236 20020917 <--
AU 2007202615 A1 20070628 AU 2007-202615 20070607 <--
PRAI US 2000-190370P P 20000317 <--
AU 2001-49263 A3 20010319 <--
EP 2001-922465 A3 20010319 <--
WO 2001-US8729 W 20010319 <--
OS MARPAT 135:267219
OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L23 ANSWER 11 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Synthesis of N-arachidonyl-O-phospho-L-serine and of N-arachidonoyl-O-phospho-L-tyrosine as nonsaturated lysophosphatic acid receptor antagonists
AB A method for preparing unsatd. antagonists of lysophosphatidic acid receptor was developed. The synthesis of both N-arachidonyl-O-phospho-L-serine (I) and N-arachidonyl-O-phospho-L-tyrosine is described. I was prepared by the reaction of L-serine with arachidonic acid in the presence of Et3N followed by the reaction of the resulting N-arachidonyl-L-serine with β-cyanoethyl phosphate.
AN 2001:601416 HCAPLUS <<LOGINID::20091001>>
DN 136:330465
TI Synthesis of N-arachidonyl-O-phospho-L-serine and of N-arachidonoyl-O-phospho-L-tyrosine as nonsaturated lysophosphatic acid receptor antagonists
AU Arsenov, D. V.; Kisel, M. A.; Strel'chenok, O. A.
CS Inst. Bioorg. Khim., NAN Belarusi, Belarus
SO Doklady Natsional'noi Akademii Nauk Belarusi (2001), 45(3), 71-73
CODEN: DNABFW; ISSN: 1561-8323
PB Belaruskaya Navuka
DT Journal
LA Russian
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L23 ANSWER 12 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Lysophosphatidic acid inhibits Ca2+ signaling in response to epidermal growth factor receptor stimulation in human astrocytoma cells by a mechanism involving phospholipase Cγ and a Gαi protein
AB The effect of the lysophospholipid mediators lysophosphatidic acid (LPA) and sphingosine 1-phosphate and the polypeptide growth factor epidermal growth factor (EGF) on the human astrocytoma cell line 1321N1 was assessed. These agonists produced a rapid and transient increase of the intracellular Ca2+ concentration When LPA was perfused before addition of EGF,

the EGF-dependent Ca2+ transient was abrogated, whereas this was not observed when EGF preceded LPA addition This inhibitory effect was not found for other EGF-mediated responses, e.g., activation of the mitogen-activated protein kinase cascade and cell proliferation, thus pointing to the existence of cross-talk between LPA and EGF for only a branch of EGF-induced responses. As 1321N1 cells expressed mRNA encoding the LPA receptors endothelial differentiation gene (Edg)-2, Edg-4, and Edg-7 and as sphingosine 1-phosphate did not interfere with LPA signaling, Edg-2, Edg-4, and/or Edg-7 could be considered as the LPA receptors mediating the aforementioned cross-talk. Attempts to address the biochem. mechanism involved in the cross-talk between the receptors were conducted by the immunopptn. approach using antibodies reacting with the EGF receptor (EGFR), phosphotyrosine, phospholipase Cγ (PLCγ)-1, and Gαi protein. LPA was found to induce coupling of PLCγ-1 to the EGFR by a mechanism involving a Gαi protein, in the absence of tyrosine phosphorylation of both PLCγ and the EGFR. These data show a cross-talk between LPA and EGF limited to a branch of EGFR-mediated signaling, which may be explained by a LPA-induced, Gαi-protein-mediated translocation of PLCγ-1 to EGFR in the absence of detectable tyrosine phosphorylation of both proteins.

AN 2000:675655 HCAPLUS <<LOGINID::20091001>>
DN 133:291530
TI Lysophosphatidic acid inhibits Ca2+ signaling in response to epidermal growth factor receptor stimulation in human astrocytoma cells by a mechanism involving phospholipase Cγ and a Gαi protein
AU Hernandez, Marita; Barrero, Maria Jose; Crespo, Mariano Sanchez; Nieto, Maria Luisa
CS Instituto de Biologia y Genetica Molecular, CSIC-Universidad de Valladolid, Valladolid, 47005, Spain
SO Journal of Neurochemistry (2000), 75(4), 1575-1582
CODEN: JONRA9; ISSN: 0022-3042
PB Lippincott Williams & Wilkins
DT Journal
LA English
OSC.G 20 THERE ARE 20 CAPLUS RECORDS THAT CITE THIS RECORD (20 CITINGS)
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 13 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Lysophosphatidic acid and sphingosine 1-phosphate stimulate endothelial cell wound healing
AB Lysophosphatidic acid (LPA) and sphingosine 1-phosphate (S1P) are potent lipid growth factors with similar abilities to stimulate cytoskeleton-based cellular functions. Their effects are mediated by a subfamily of G protein-coupled receptors (GPCRs) encoded by endothelial differentiation genes (edgs). The authors hypothesize that large quantities of LPA and S1P generated by activated platelets may influence endothelial cell functions. Using an in vitro wound healing assay, the authors observed that LPA and S1P stimulated closure of wounded monolayers of human umbilical vein endothelial cells and adult bovine aortic endothelial cells, which express LPA receptor Edg2, and S1P receptors Edg1 and Edg3. The two major components of wound healing, cell migration and proliferation, were stimulated individually by both lipids. LPA and S1P also stimulated intracellular Ca2+ mobilization and mitogen-activated protein kinase (MAPK) phosphorylation. Pertussis toxin partially blocked the effects of both lipids on endothelial cell migration, MAPK phosphorylation, and Ca2+ mobilization, implicating Gi/o-coupled Edg receptor signaling in endothelial cells. LPA and S1P did not cross-desensitize each other in Ca2+ responses, suggesting involvement of distinct receptors. Thus LPA and S1P affect endothelial cell functions

through signaling pathways activated by distinct GPCRs and may contribute to the healing of wounded vasculatures.

AN 2000:189961 HCAPLUS <<LOGINID::20091001>>
DN 132:320462
TI Lysophosphatidic acid and sphingosine 1-phosphate stimulate endothelial cell wound healing
AU Lee, Hsinyu; Goetzl, Edward J.; An, Songzhu
CS Department of Medicine, University of California Medical Center, San Francisco, CA, 94143-0711, USA
SO American Journal of Physiology (2000), 278(3, Pt. 1), C612-C618
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
OSC.G 122 THERE ARE 122 CAPLUS RECORDS THAT CITE THIS RECORD (122 CITINGS)
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 14 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Methods using a lysophosphatidic acid receptor agonist for promoting survival of myelin-producing cells
AB The invention is in the field of neurobiol., and relates particularly to methods useful for enhancing the survival of myelin producing cells, in particular Schwann cells and oligodendrocytes, and thereby to treating diseases of the nervous system involving loss of myelination or aberrant myelination. The methodol. of the invention uses a survival-promoting amount of an lysophosphatidic acid (LPA) receptor agonist, e.g. LPA.
AN 2000:133529 HCAPLUS <<LOGINID::20091001>>
DN 132:175856
TI Methods using a lysophosphatidic acid receptor agonist for promoting survival of myelin-producing cells
IN Chun, Jerold J. M.; Weiner, Joshua A.; Wickens, Philip L.; Begleiter, Leath E.
PA The Regents of the University of California, USA; Allelix Biopharmaceuticals Inc.
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000009139	A2	20000224	WO 1999-US18069	19990810 <--
	WO 2000009139	A3	20000518		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6150345	A	20001121	US 1998-153464	19980915 <--
	AU 9954735	A	20000306	AU 1999-54735	19990810 <--
PRAI	US 1998-96008P	P	19980810 <--		
	US 1998-96924P	P	19980818 <--		
	US 1998-153464	A	19980915 <--		
	WO 1999-US18069	W	19990810 <--		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OSC.G 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L23 ANSWER 15 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Enhancement of the Migration of Metastatic Human Breast Cancer Cells by Phosphatidic Acid
AB Phosphatidic acid (PA), lysophosphatidic acid (LPA), and sphingosine 1-phosphate (SPP) are naturally occurring phospholipids which induce a variety of effects as extracellular messengers. In this study, we compared the effects of these phospholipid signaling mols. on the migration of invasive and noninvasive breast cancer cell lines, an index of the metastatic potential of these cells. As previously demonstrated, invasive MDA-MB-231 breast cancer cells exhibited increased constitutive (nonstimulated) migration in comparison to poorly invasive MCF-7 cells. Phosphatidic acid employed at nanomolar concns. markedly potentiated migration of the invasive cells but had no effect on migration of either the noninvasive MCF-7 cells or nonneoplastic human epithelial cells. Lysophosphatidic acid and sphingosine 1-phosphate inhibited both the directed (chemotactic) and random (chemokinetic) migration of MDA-MB-231 cells. Expts. were undertaken to characterize the signaling pathway involved in constitutive and PA-stimulated migration of MDA-MB-231 cells. The tyrosine kinase inhibitors staurosporine and genistein inhibited constitutive and PA-induced migration in a dose-dependent manner, consistent with a role for tyrosine phosphorylation in the migratory response. In addition, the phosphatidylinositol (PI) 3' kinase inhibitors wortmannin and LY294002 strongly inhibited both the constitutive and PA-stimulated migration of the invasive breast cancer cells, indicating that PI-3' kinase plays an important role in the metastatic migration of breast cancer cells. Finally, PA-induced migration of MDA-MB-231 was markedly attenuated by pretreatment of cells with Clostridium difficile Toxin B, pertussis toxin and suramin, implying a role for a Gi receptor-dependent process involving activation of the small GTP-binding protein Rho. Since an enhanced ability to migrate heightens the metastatic potential of cells within solid tumors, our results suggest that the metastatic capabilities of breast cancer cells may be enhanced by a receptor-driven cellular process initiated by phosphatidic acid or related lipid phosphate messengers. (c) 2000 Academic Press.
AN 2000:115022 HCAPLUS <<LOGINID::20091001>>
DN 132:263313
TI Enhancement of the Migration of Metastatic Human Breast Cancer Cells by Phosphatidic Acid
AU Sliva, Daniel; Mason, Rebekah; Xiao, Hongyan; English, Denis
CS Experimental Cell Research Program, Methodist Research Institute, Clarian Health Partners Inc., Indianapolis, IN, 46202, USA
SO Biochemical and Biophysical Research Communications (2000), 268(2), 471-479
CODEN: BBRCA9; ISSN: 0006-291X
PB Academic Press
DT Journal
LA English
OSC.G 33 THERE ARE 33 CAPLUS RECORDS THAT CITE THIS RECORD (33 CITINGS)
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 16 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Lysophosphatidic acid-induced Ca2+ mobilization in the neural retina of chick embryo
AB Lysophosphatidic acid (LPA) plays various roles in the regulation of cell growth as a lipid mediator. We studied the effect of LPA on intracellular Ca2+ concentration ([Ca2+]i) with Fura-2 in the neural retina of chick embryo during neurogenesis. Bath application of LPA (1-100 μM) to the embryonic day 3 (E3) chick retina caused an increase in [Ca2+]i in a dose-dependent manner, with an EC50 value of 9.2 μM. The Ca2+ rise was

also evoked in a Ca2+-free medium, suggesting that release of Ca2+ from intracellular Ca2+ stores (Ca2+ mobilization) was induced by LPA. U-73122, a blocker of phospholipase C (PLC), inhibited the Ca2+ rise to LPA. Pertussis toxin partially inhibited the Ca2+ rise to LPA, indicating that Gi/Go protein was at least partially involved in the LPA response. The developmental profile of the LPA response was studied from E3 to E13. The Ca2+ rise to LPA declined drastically from E3 to E7, in parallel with decrease in mitotic activity of retinal progenitor cells. The signal transduction pathway and developmental profile of the Ca2+ response to LPA were the same as those of the Ca2+ response to ATP, which enhances the proliferation of retinal progenitor cells. The coapplication of LPA with ATP resulted in enhancement of Ca2+ rise in the E3 chick retina. Our results show that LPA induces Ca2+ mobilization in the embryonic chick retina during neurogenesis.

AN 2000:14366 HCAPLUS <<LOGINID::20091001>>
DN 132:163687
TI Lysophosphatidic acid-induced Ca2+ mobilization in the neural retina of chick embryo
AU Zhou, Wen-Liang; Sugioka, Miho; Yamashita, Masayuki
CS Department of Physiology, Osaka University Medical School, Suita, 565-0871, Japan
SO Journal of Neurobiology (1999), 41(4), 495-504
CODEN: JNEUBZ; ISSN: 0022-3034
PB John Wiley & Sons, Inc.
DT Journal
LA English
OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 17 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI A metabolic path for the degradation of lysophosphatidic acid, an inhibitor of lysophosphatidylcholine lysophospholipase, in neuronal nuclei of cerebral cortex
AB Neuronal nuclei isolated from rabbit cerebral cortex were found to be enriched in an NEM-insensitive lysophosphatidic acid (lysoPA) phosphohydrolase activity. LysoPA is an inhibitor of the nuclear lysophosphatidylcholine (lysoPC) lysophospholipase, and by preserving lysoPC levels, lysoPA boosted the nuclear production of the acyl analog of platelet-activating factor by promoting the acetylation of lysoPC (Baker, R. R.; Chang, H.-y., 1999). The nuclear phosphohydrolase converts lysoPA to 1-monoacylglycerol, and thus eliminates this lysoPA inhibition of lysoPC lysophospholipase. The nuclear lysoPA phosphohydrolase specific activity was more than three times that observed for the nuclear lysoPA lysophospholipase (Baker, R. R.; Chang, H.-y., 1999), and represents a more active route for nuclear lysoPA removal. The neuronal nuclear lysoPA phosphohydrolase was inhibited at acidic pH, and also inhibited by calcium ions. The 1-monoacylglycerol product of the phosphohydrolase is rapidly degraded by neuronal monoacylglycerol lipase, an enzyme some sevenfold more active than the phosphohydrolase and sensitive to inhibition by arachidonoyl trifluoromethyl ketone (AACOCF3). Both acidic pH and free fatty acid inhibited the lipase. In the absence of AACOCF3, production of fatty acid from lysoPA substrate could be largely attributed to the sequential actions of the nuclear phosphohydrolase and lipase. This facilitates fatty acid recycling back into phospholipid by lysophospholipid acylation when ATP levels are restored following periods of brain ischemia. At relatively low concns., sphingosine-1-phosphate, and alkylglycerophosphate were the most effective phosphohydrolase inhibitors while phosphatidic acid, alkylacetylglycerophosphate and ceramide were without effect. LysoPA is an interesting regulatory mol. that can potentially preserve

lysophosphatidylcholine within the nuclear membrane for use in acetylation reactions. Thus conditions relevant to brain ischemia such as falling pH, falling ATP concns., rising fatty acid and intracellular calcium levels may, by slowing this metabolic path for lysoPA loss, promote the production of acyl PAF and contribute to the increased levels of the acetylated lipids noted in ischemia.

AN 1999:793729 HCAPLUS <<LOGINID::20091001>>
DN 132:149456
TI A metabolic path for the degradation of lysophosphatidic acid, an inhibitor of lysophosphatidylcholine lysophospholipase, in neuronal nuclei of cerebral cortex
AU Baker, R. R.; Chang, H.-y.
CS Department of Biochemistry, University of Toronto, Toronto, ON, Can.
SO Biochimica et Biophysica Acta, Molecular and Cell Biology of Lipids (2000), 1483(1), 58-68
CODEN: BBMLFG; ISSN: 1388-1981
PB Elsevier B.V.
DT Journal
LA English
OSC.G 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 18 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI In vitro autoradiographic visualization of guanosine-5'-O-(3-[35S]thio)triphosphate binding stimulated by sphingosine 1-phosphate and lysophosphatidic acid
AB Sphingosine 1-phosphate or lysophosphatidic acid activation of guanosine-5'-O-(3-[35S]thio)-triphosphate ([35S]GTPγS) binding to G proteins was studied by in vitro autoradiog. in rat and guinea pig brain. The highest stimulation of [35S]GTPγS binding by sphingosine 1-phosphate was observed in the mol. layer of the cerebellum. Marked stimulation was observed in most forebrain areas, including neocortex and striatum. With the exception of the substantia gelatinosa and nucleus of the solitary tract, sphingosine 1-phosphate-enhanced binding was weaker in the brainstem and spinal cord. Lysophosphatidic acid-enhanced labeling was only observed in white matter areas. The G protein inhibitor 5'-p-fluorosulfonylbenzoyl guanosine completely inhibited lysophosphatidic acid-enhanced [35S]GTPγS binding but only partially sphingosine 1-phosphate-enhanced binding. N-Ethylmaleimide abolished binding stimulated by both agonists. Sphingosine 1-phosphate enhanced labeling by another GTP analog (β,γ-imido[8-3H]guanosine-5'-triphosphate) similarly to that of [35S]GTPγS. Lysophosphatidic acid stimulated [35S]GTPγS binding in the olfactory bulb, glia limitans, and cortical subventricular zone of 1-day-old rats, whereas enhanced labeling was not observed in the latter area of 5-day-old rats. Sphingosine 1-phosphate stimulated binding in the cortical and striatal subventricular zones and olfactory bulb in 1- and 5-day-old rats. In the absence of radioligand for sphingosine 1-phosphate and lysophosphatidic acid receptors, [35S]GTPγS autoradiog. provides a unique opportunity to study the spatial distribution, ontogeny, and coupling properties of these receptors.

AN 1999:547170 HCAPLUS <<LOGINID::20091001>>
DN 131:283533
TI In vitro autoradiographic visualization of guanosine-5'-O-(3-[35S]thio)triphosphate binding stimulated by sphingosine 1-phosphate and lysophosphatidic acid
AU Waeber, Christian; Chiu, Mary L.
CS Massachusetts General Hospital and Harvard Medical School, Charlestown, MA, 02129, USA

SO Journal of Neurochemistry (1999), 73(3), 1212-1221
CODEN: JONRA9; ISSN: 0022-3042
PB Lippincott Williams & Wilkins
DT Journal
LA English
OSC.G 16 THERE ARE 16 CAPLUS RECORDS THAT CITE THIS RECORD (16 CITINGS)
RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 19 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Influence of pertussis toxin on local progression and metastasis after orthotopic implantation of the human prostate cancer cell line PC-3 in nude mice
AB Tumor cell migration is a fundamental process of metastasis. Pertussis toxin inhibits lysophosphatidic acid-related cell migration by ADP-ribosylation of G proteins. The authors examined the influence of pertussis toxin (PTX) on the progression and metastasis of the human hormone-insensitive prostate cancer cell line PC-3 after orthotopic implantation in nude mice. In 30 athymic male nude mice (NMRI), 5 + 105 PC-3 cells were injected into the dorsal prostate. After 7 d, 15 mice received a total of 6 i.p. injections of 5 μg PTX/100 g at an interval of 4 d. The other 15 mice received phosphate-buffered saline and served as control. All mice were killed at 37 d followed by macroscopical and histol. evaluation of local tumor growth and metastasis. In the control group, tumorigenicity was 100% (15 out of 15). Mean weight of the tumor-bearing unit of prostate and seminal vesicles was 541 mg (243-763 mg). PTX following orthotopic implantation of the human hormone-insensitive PC-3 cell line significantly reduces local tumor growth as well as metastasis to loco-regional lymph nodes.
AN 1999:145092 HCAPLUS <<LOGINID::20091001>>
DN 130:333942
TI Influence of pertussis toxin on local progression and metastasis after orthotopic implantation of the human prostate cancer cell line PC-3 in nude mice
AU Bex, A.; Lummen, G.; Rembrink, K.; Otto, T.; Metz, K.; Rubben, H.
CS Clinic of Urology, University of Essen Medical School, Essen, Germany
SO Prostate Cancer and Prostatic Diseases (1999), 2(1), 36-40
CODEN: PCPDFW; ISSN: 1365-7852
PB Stockton Press
DT Journal
LA English
OSC.G 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 20 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Actin depolymerizing factor and cofilin phosphorylation dynamics: response to signals that regulate neurite extension
AB The actin assembly-regulating activity of actin depolymg. factor (ADF)/cofilin is inhibited by phosphorylation. Studies were undertaken to characterize the signaling pathways and phosphatases involved in activating phosphorylated ADF (pADF), emphasizing signals related to neuronal process extension. Western blots using antibodies to ADF and cofilin, as well as an ADF/cofilin phosphoepitope-specific antibody characterized in this paper, were used to measure changes in the phosphorylation state and phosphate turnover of ADF/cofilin in response to inhibitors and agents known to influence growth cone motility. Increases in both [Ca2+]i and cAMP levels induced rapid pADF dephosphorylation in HT4 and cortical neurons. Calcium-dependent dephosphorylation depended on the activation of protein phosphatase 2B

(PP2B), while cAMP-dependent dephosphorylation was likely through activation of PP1. Growth factors such as NGF and insulin also induced rapid pADF/pcofilin dephosphorylation, with NGF-stimulated dephosphorylation in PC12 cells correlated with the translocation of ADF/cofilin to ruffling membranes. Of special interest was the finding that the rate of phosphate turnover on both pADF and pcofilin could be enhanced by growth factors without changing net pADF levels, demonstrating that growth factors can activate bifurcating pathways that promote both phosphorylation and dephosphorylation of ADF/cofilin. All exptl. results indicated that dynamics of phosphorylation on ADF and cofilin are coordinately regulated. Signals that decreased pADF levels are associated with increased process extension, while agents that increased pADF levels, such as lysophosphatidic acid, inhibit process extension. These data indicate that dephosphorylation/activation of pADF is a significant response to the activation of signal pathways that regulate actin dynamics and alter cell morphol. and neuronal outgrowth.

AN 1998:110981 HCAPLUS <<LOGINID::20091001>>
DN 128:215788
OREF 128:42709a,42712a
TI Actin depolymerizing factor and cofilin phosphorylation dynamics: response to signals that regulate neurite extension
AU Meberg, Peter J.; Ono, Shoichiro; Minamide, Laurie S.; Takahashi, Masami; Bamburg, James R.
CS Department of Biochemistry and Molecular Biology, Colorado State University, Fort Collins, CO, 80523-1870, USA
SO Cell Motility and the Cytoskeleton (1998), 39(2), 172-190
CODEN: CMCYEO; ISSN: 0886-1544
PB Wiley-Liss, Inc.
DT Journal
LA English
OSC.G 127 THERE ARE 127 CAPLUS RECORDS THAT CITE THIS RECORD (127 CITINGS)
RE.CNT 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 21 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Structure/activity relationships in lysophosphatidic acid: the 2-hydroxyl moiety
AB Although lipid phosphoric acid mediators such as lysophosphatidic acid (LPA) are now recognized widely as intercellular signaling mols., the medicinal chemical of these mediators is poorly developed. With the goal of achieving a better understanding of the structure activity relationships in LPA, we have synthesized and tested a series of LPA analogs that lack the 2-hydroxyl moiety. Our series consisted of compds. with 2, 3, or 4 carbon diol or amino alc. backbones and oleoyl or palmitoleoyl acyl groups. These mols. cannot be acylated further to form phosphatidic acids, nor do they have chiral centers. The rank order potency of these compds. in mobilization of calcium in MDA MB-231 cells suggested a maximum optimal chain length of 24-25 atoms. However, high potency for the inhibition of adenylyl cyclase in these cells was achieved only by one compound that also contained a dissociable proton five bond lengths from the phosphorus atom. That compound, N-oleoyl-2-hydroxyethyl-1-phosphate, was nearly equipotent to 1-oleoyl LPA in both assays. The striking mimicry of LPA by the ethanolamine-based compound and the presence of fatty acid amides in tissue prompts us to propose that phosphorylated N-acyl ethanolamides occur naturally.
AN 1997:459506 HCAPLUS <<LOGINID::20091001>>
DN 127:174436
OREF 127:33753a,33756a
TI Structure/activity relationships in lysophosphatidic acid: the 2-hydroxyl

moiety
AU Lynch, Kevin R.; Hopper, Darrin W.; Carlisle, Steven J.; Catalano, John G.; Zhang, Ming; Macdonald, Timothy L.
CS Department of Pharmacology, University of Virginia, Charlottesville, VA, 22908, USA
SO Molecular Pharmacology (1997), 52(1), 75-81
CODEN: MOPMA3; ISSN: 0026-895X
PB Williams & Wilkins
DT Journal
LA English
OSC.G 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)

L23 ANSWER 22 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Characterization of sphingosine 1-phosphate-induced actions and its signaling pathways in rat hepatocytes
AB Sphingosine 1-phosphate (S-1-P) and lysophosphatidic acid (LPA) stimulated glycogen phosphorylase, a rate-limiting enzyme responsible for glycogenolysis, in association with Ca2+ mobilization and phospholipase C (PLC) activation in rat hepatocytes. S-1-P, but not LPA, also inhibited adenosine 3',5'-cyclic monophosphate accumulation reflecting adenylyl cyclase inhibition. S-1-P-induced PLC activation, Ca2+ mobilization, and phosphorylase activation were markedly enhanced by primary culture of the cells for 24 h, whereas the inhibitory adenosine 3',5'-cyclic monophosphate response was unchanged by increasing culture time. Activation of the PLC-Ca2+ system during primary culture was specific to the lysosphingolipid; PLC and Ca2+ responses to LPA and NaF were unchanged or slightly attenuated by increasing culture time. Pertussis toxin treatment almost completely suppressed the S-1-P-induced inhibition of adenylyl cyclase but hardly influenced the lipid-induced activation of PLC and its cascade reactions. It is concluded that S-1-P, through an LPA receptor-independent mechanism, stimulates two signaling pathways, i.e., activation of the PLC-Ca2+ system and inhibition of adenylyl cyclase, through distinct S-1-P receptor-transducer systems, resulting in the modulation of glycogenolysis in rat hepatocytes.
AN 1997:354979 HCAPLUS <<LOGINID::20091001>>
DN 127:79075
OREF 127:15101a,15104a
TI Characterization of sphingosine 1-phosphate-induced actions and its signaling pathways in rat hepatocytes
AU Im, Dong-Soon; Fujioka, Toshiyuki; Katada, Toshiaki; Kondo, Yoichi; Ui, Michio; Okajima, Fumikazu
CS Lab. Signal Transduction, Inst. Mol. Cellular Regulation, Gunma Univ., Maebashi, 371, Japan
SO American Journal of Physiology (1997), 272(5, Pt. 1), G1091-G1099
CODEN: AJPHAP; ISSN: 0002-9513
PB American Physiological Society
DT Journal
LA English
OSC.G 40 THERE ARE 40 CAPLUS RECORDS THAT CITE THIS RECORD (40 CITINGS)
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 23 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI N-palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids are selective competitive antagonists of the lysophosphatidic acid receptors
AB Lysophosphatidic acid is best characterized as member of a lipid mediator family with growth factor-like activities that act through a class of G protein-coupled plasma membrane receptors. In Xenopus Levis oocytes, lysophosphatidate activates at least two pharmacol. distinct receptor

subtypes distinguished by 1-acyl-sn-glycero-2,3-cyclic phosphate . Both of these naturally occurring ligands elicit oscillatory Cl- currents in the oocyte through G protein-coupled activation of the phosphoinositide/Ca2+ second messenger system, which in turn leads to the opening of Ca2+ -activated Cl- channels. We developed an improved chemical synthesis and purification procedure for two N-acylated amino acid phosphates. N-Palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids inhibited the lysophosphatidate-activated Cl- currents with IC50 values of 5.4 and 6.5 nM at the high affinity site and 805 and 172 nM at the low affinity receptor site, resp. In selective activation of the cyclic lysophosphatidate receptor, IC50 values of 330 and 490 nM were obtained, resp. The D- and L-stereoisomers were equally effective when applied extracellularly. In contrast, they were ineffective when microinjected into the oocyte, indicating an extracellular site of inhibition. The inhibitors did not alter currents elicited by the different acetylcholine, serotonin, and glutamate receptors expressed heterologously in the oocyte. Pharmacol. anal. of the results indicates that N-palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids are potent and specific competitive inhibitors of the lysophosphatidate receptors in the X. Levis oocyte.

AN 1996:570299 HCAPLUS <<LOGINID::20091001>>
DN 125:265929
OREF 125:49377a,49380a
TI N-palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids are selective competitive antagonists of the lysophosphatidic acid receptors
AU Liliom, Karoly; Bittman, Robert; Swords, Bernadette; Tigyi, Gabor
CS Department of Physiology and Biophysics, University of Tennessee, Memphis, TN, 38163, USA
SO Molecular Pharmacology (1996), 50(3), 616-623
CODEN: MOPMA3; ISSN: 0026-895X
PB Williams & Wilkins
DT Journal
LA English
OSC.G 56 THERE ARE 56 CAPLUS RECORDS THAT CITE THIS RECORD (56 CITINGS)

L23 ANSWER 24 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Inhibitors of lipid phosphatidate receptors: N-palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids
AB An improved synthesis of two lipid phosphoric acids, N-palmitoyl-L-serinephosphoric acid (NP-Ser-PA) and N-palmitoyl-L-tyrosinephosphoric acid (NP-Tyr-PA), from L-serine and L-tyrosine benzyl esters is described. The sequence of N-acylation, followed by phosphitylation with dibenzyl N,N-diisopropylphosphoramidite, oxidation to the corresponding phosphate triesters, and simultaneous debenzylation of the dibenzyl phosphate and benzyl carboxylic esters gave NP-Ser-PA and NP-Tyr-PA in high overall yields. NP-Ser-PA and NP-Tyr-PA and their D-stereoisomers were potent reversible inhibitors of the lysophosphatidic acid receptors expressed in Xenopus oocytes, thus providing prototypic structures for the development of inhibitors of the lysophosphitidate family of phospholipid growth factors.
AN 1996:154614 HCAPLUS <<LOGINID::20091001>>
DN 124:317814
OREF 124:58961a,58964a
TI Inhibitors of lipid phosphatidate receptors: N-palmitoyl-serine and N-palmitoyl-tyrosine phosphoric acids
AU Bittman, Robert; Swords, Bernadette; Liliom, Karoly; Tigyi, Gabor
CS Dep. Chemistry Biochemistry, Queens College City Univ. New York, Flushing, NY, 11367-1597, USA
SO Journal of Lipid Research (1996), 37(2), 391-8

CODEN: JLPRAW; ISSN: 0022-2275
PB Lipid Research, Inc.
DT Journal
LA English
OSC.G 24 THERE ARE 24 CAPLUS RECORDS THAT CITE THIS RECORD (24 CITINGS)

L23 ANSWER 25 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Selective inhibition of DNA polymerase-α family with chemically synthesized derivatives of PHYLPA, a unique Physarum lysophosphatidic acid
AB PHYLPA, a unique Physarum lysophosphatidic acid (LPA), showed selective inhibition of a family of DNA polymerase α, including DNA polymerases α, δ and ε; but no inhibition of DNA polymerase β or γ was observed To reveal the mol. mechanism of inhibition of DNA polymerases by PHYLPA, four stereoisomers and some other derivs. were synthesized and their effects on DNA polymerases were studied. Among eight derivs. synthesized, PHYLPA-1 (the natural PHYLPA; sodium 1-O-[(9'S,10'R)-9',10'-methanohexadecanoyl]-sn-glycerol 2,3-cyclic phosphate) and PHYLPA-2 (sodium 3-O-[(9'S,10'R)-9',10'-methanohexadecanoyl]-sn-glycerol 1,2-cyclic phosphate) were strong and specific inhibitors of a family of DNA polymerase α. But their stereoisomers PHYLPA-3 (sodium 1-O-[(9'R,10'S)-9',10'-methanohexadecanoyl]-sn-glycerol 2,3-cyclic phosphate) and PHYLPA-4 (sodium 3-O-[(9'R,10'S)-9',10'-methanohexadecanoyl]-sn-glycerol 1,2-cyclic phosphate) were weak inhibitors, showing the critical importance of stereochem. of a cyclopropane-containing fatty acid for the inhibitory activity. Some derivs. having no cyclopropane-containing fatty acids - palmitoyl-, oleoyl-, and palmitoleoyl-PHYLPA -showed inhibition to some extent; but 1-palmitoyl and 1-oleoyl lysophosphatidic acid, which has no cyclic phosphate, did not show an apparent inhibitor activity on DNA polymerases. Hence, the extent of the inhibition apparently depends on the stereochem. of both the fatty acid moiety and the cyclic phosphate.
AN 1995:764871 HCAPLUS <<LOGINID::20091001>>
DN 123:221508
OREF 123:39331a,39334a
TI Selective inhibition of DNA polymerase-α family with chemically synthesized derivatives of PHYLPA, a unique Physarum lysophosphatidic acid
AU Murakami-Murofushi, Kimiko; Kobayashi, Susumu; Onimura, Kenjiro; Matsumoto, Miyoko; Shioda, Masaki; Yoshida, Shonen; Shoji, Mami; Murofushi, Hiromu
CS Department of Biology, Faculty of Science, Ochanomizu University, Ohtsuka 2-1-1, Bunkyo-ku, Tokyo, 112, Japan
SO Biochimica et Biophysica Acta, Lipids and Lipid Metabolism (1995), 1258(1), 57-60
CODEN: BBLLA6; ISSN: 0925-4439
PB Elsevier B.V.
DT Journal
LA English
OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

L23 ANSWER 26 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Use of photoreactive substrates for characterization of lysophosphatidate acyltransferases from developing soybean cotyledons
AB Photoreactive lipid analogs, namely, 1-acyl-2-(12-azidooleoyl)glycero-3-phosphocholine (N3-PC) and 1-acyl-2-(12-azidooleoyl)glycero-3-phosphoethanolamine (N3-PE) have been synthesized as previously described [R. Rajasekharan and J. D. Kemp (1994) J. Lipid Res. 35, 45-51]. Azidophosphatidic acid was produced by hydrolyzing N3-PC with phospholipase D. All of the lysophospholipid analogs, 2-(12-azidooleoyl)glycero-3-phosphate (N3-LPA),

2-(12-azidooleoyl)glycero-3-phosphocholine (N3-LPC), and 2-(12-azidooleoyl)glycero-3-phosphoethanolamine (N3-LPE), were produced from appropriate azidophospholipids by lipase treatment. The photoactive lysophospholipid analogs were recognized as substrates by acyltransferases in the dark and as irreversible inhibitors after photolysis with UV light. The photoinactivation of acyltransferases by azidolysophospholipids was protected by the addition of natural lysophospholipids. Incubation of developing soybean microsomal membranes with N3-LPA followed by photolysis resulted in 69% inhibition of lysophosphatidic acid (LPA) acyltransferase and also had significant inhibitory effects on lysophosphatidylcholine (LPC) and lysophosphatidylethanolamine (LPE) acyltransferases, indicating that the LPA analog interacts with all the lysophospholipid acyltransferases. When the membranes were photolyzed with N3-LPC or N3-LPE and assayed, the membranes showed approx. 50% inactivation of LPC and LPE acyltransferase activities, whereas LPA acyltransferase was unaffected, suggesting that a single enzyme might acylate both LPC and LPE. The recognition of these photoreactive lipid analogs by acyltransferases will facilitate the identification and purification of these membrane-bound enzymes.

AN 1994:599203 HCAPLUS <<LOGINID::20091001>>
DN 121:199203
OREF 121:36094h,36095a
TI Use of photoreactive substrates for characterization of lysophosphatidate acyltransferases from developing soybean cotyledons
AU Rajasekharan, Ram; Nachiappan, Vasanthi
CS Plant Genetic Eng. Lab., New Mexico State Univ., Las Cruces, NM, 88003, USA
SO Archives of Biochemistry and Biophysics (1994), 311(2), 389-94
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA English
OSC.G 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)

L23 ANSWER 27 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Phosphatidic acid, lysophosphatidic acid, and lipid A are inhibitors of glycosylphosphatidylinositol-specific phospholipase D. Specific inhibition of a phospholipase by product analogs?
AB Previous work has suggested that the glycosylphosphatidylinositol-specific phospholipase D (GPI-PLD) purified from bovine serum is inhibited by phosphatdic acid (PA). In this study, the specificity and mechanism of this phenomenon using [3H]myristate-labeled variant surface glycoprotein dispersed in Nonidet P-40 as substrate is reported. Inhibition of GPI-PLD by PAs (IC50 .apprx.1 μM) was relatively independent of the length or degree of unsatn. of the fatty acyl chains. It was also observed that lysophosphatidic acid and several natural and synthetic lipid A prepns. were inhibitory in the same concentration range. The inhibitory potency of PA, lysophosphatidic acid, and lipid A was dependent on the detergent concentration in the assay but in all cases this was in a large (i.e. > 100-fold) molar excess over the inhibitor. The inhibitory lipids did not affect substrate availability nor did they reduce hydrolysis of variant surface glycoprotein by a bacterial phosphatidylinositol-specific phoshpolipase C. Studies with a wide range of other lipids, detergents, and phosphate esters indicated that inhibition was specific for lipids containing a phosphomonoester group. The data suggest that inhibition is due to a direct interaction between PA (or lipid A) and the GPI-PLD rather than an indirect effect on the substrate particle.

AN 1993:250409 HCAPLUS <<LOGINID::20091001>>
DN 118:250409
OREF 118:43347a,43350a

TI Phosphatidic acid, lysophosphatidic acid, and lipid A are inhibitors of glycosylphosphatidylinositol-specific phospholipase D. Specific inhibition of a phospholipase by product analogs?
AU Low, Martin G.; Huang, Kuo Sen
CS Coll. Physicians Surg., Columbia Univ., New York, NY, 10032, USA
SO Journal of Biological Chemistry (1993), 268(12), 8480-90
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
OSC.G 17 THERE ARE 17 CAPLUS RECORDS THAT CITE THIS RECORD (17 CITINGS)

L23 ANSWER 28 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Inhibition of eukaryotic DNA polymerase α with a novel lysophosphatidic acid (PHYLPA) isolated from myxoamebae of Physarum polycephalum
AB A specific inhibitor of DNA polymerase-α was isolated from the lipid fraction prepared from myxoamoebae of a true slime mold, P. polycephalum. The purified substance was subjected to structural studies by fast atom bombardment mass spectroscopy, IR spectroscopy, and 2-dimensional NMR spectroscopy. The structure of this substance was thereby suggested to be a novel lysophosphatidic acid (LPA) composed of cyclic phosphate and cyclopropane-containing hexadecanoic acid. This substance was named PHYLPA (Physarum LPA). PHYLPA inhibited >80% of affinity-purified calf thymus I activity at a concentration of 10 μg/mL (.apprx.20 μM). Inhibition was observed for I but not for DNA polymerase-β or from various eukaryotic species, nor did it inhibit DNA polymerase I from E. coli. From kinetic analyses, the inhibition was considered to be caused by the interaction of PHYLPA with template DNA.
AN 1992:607756 HCAPLUS <<LOGINID::20091001>>
DN 117:207756
OREF 117:35761a,35764a
TI Inhibition of eukaryotic DNA polymerase α with a novel lysophosphatidic acid (PHYLPA) isolated from myxoamebae of Physarum polycephalum
AU Murakami-Murofushi, Kimiko; Shioda, Masaki; Kaji, Kazuhiko; Yoshida, Shonen; Murofushi, Hiromu
CS Fac. Sci., Ochanomizu Univ., Tokyo, 112, Japan
SO Journal of Biological Chemistry (1992), 267(30), 21512-17
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
OSC.G 33 THERE ARE 33 CAPLUS RECORDS THAT CITE THIS RECORD (34 CITINGS)

L23 ANSWER 29 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Characterization of dolichol and dolichyl phosphate phosphatase from soya beans (Glycine max)
AB A series of polyprenols, ranging in length from 15 to 22 isoprene units, has been isolated from soybeans (Glycine max) and purified by high-pressure liquid chromatog. NMR, IR, and mass spectra of the compds. indicated that they are α-saturated polyprenols of the dolichol type. The amount present in dry seeds was .apprx. 9 mg/100 g, whereas dolichyl phosphate (Dol-P) was present only in trace amts. Dol-P phosphatase (DPP) activity was detected in the microsomal fraction of 5-day-old germinating soybean cotyledons. The DPP activity was linear with respect to time and protein concentration and exhibited a broad pH optimum (pH 7-9). Triton X-100 was necessary for significant enzyme activity. Enzyme activity was slightly enhanced by EDTA, whereas dithiothreitol was without effect. An apparent Km of 5 μM was determined for Dol-P. Bivalent metal ions were not required for enzyme activity. A number of phosphorylated compds. tested as enzyme substrates (including a number of nucleoside

phosphates, glucose 6-phosphate, Na β-glycerophosphate, and Na4P2O7) did not compete with [1-3H]Dol-P as substrate. A number of phospholipids were also tested for their ability to act as DPP substrates. At 1 mM concentration, phosphatidylcholine, phosphatidylethanolamine, phosphatidic acid, and lysophosphatidic acid each exhibited enzymic activity. However, at 0.1 mM concentration, phosphatidylcholine and phosphatidylethanolamine were slightly stimulatory, whereas phosphatidic acid and lysophosphatidic acid were still inhibitory. Phosphatidic acid showed competitive inhibition.

AN 1983:485171 HCAPLUS <<LOGINID::20091001>>
DN 99:85171
OREF 99:13097a,13100a
TI Characterization of dolichol and dolichyl phosphate phosphatase from soya beans (Glycine max)
AU Ravi, Kothapalli; Rip, Jack W.; Carroll, Kenneth K.
CS Dep. Biochem., Univ. West. Ontario, London, ON, N6A 5C1, Can.
SO Biochemical Journal (1983), 213(2), 513-18
CODEN: BIJOAK; ISSN: 0306-3275
DT Journal
LA English
OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

L23 ANSWER 30 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Pulmonary phosphatidic acid phosphatase. A comparative study of the aqueously dispersed phosphatidate-dependent and membrane-bound phosphatidate-dependent phosphatidic acid phosphatase activities of rat lung
AB The properties of the aqueously dispersed phosphatidate-dependent phosphatidate phosphatase (EC 3.1.3.4) (I) activities of rat lung were studied in microsomal and cytosol prepns. and compared with those of the membrane-bound phosphatidate-dependent activities. Microsomal I displayed a prominent pH optimum at 6.5 with a minor peak which varied between 7.5-8 in different expts. The major cytosol I activity was at the higher pH (7.5-8.0) but a distinct optimum was also observed at pH 6.0-6.5. With the membrane-bound substrate, a single broad optimum was observed between pH 7.4 and 8.0 with the cytosol and 6.5-7.5 with the microsomal fraction. Subcellular fractionation studies revealed that the microsomal fraction possessed the greatest proportion of the total I activity and the highest relative specific activity. However, studies with marker enzymes indicated that the aqueously dispersed phosphatidate-dependent activity could be present in plasma membrane, lysosomes, and osmiophilic lamellar bodies as well as in the endoplasmic reticulum. The aqueously dispersed phosphatidic acid-dependent activities present in the microsomal and supernatant fractions were inhibited by Ca2+, Mn2+, F-, and by high concns. of Mg2+. In contrast to the membrane-bound phosphatidate-dependent activities, there was little Mg2+ stimulation and only a very slight inhibitory effect was noted with EDTA. A small EDTA-dependent Mg2+ stimulation could be observed with the microsomal fraction but only at the lower pH optimum (6.5). The presence of a number of phosphate esters tended to stimulate rather than inhibit the microsomal activity, indicating that I is relatively specific for lipid substrates. Marked inhibitions were noted with lysophosphatidic acid and phosphatidylglycerol phosphate. Phosphatidylcholine produced a slight inhibition. The results indicate that the bulk of the aqueously dispersed phosphatidate-dependent I activities of rat lung microsomes and cytosol is not related to the activities observed with membrane-bound phosphatidate. The Mg2+-dependent I activities may be synonymous. However, unequivocal conclusions will only be possible when the polypeptide or polypeptides responsible for these activities can be purified.

AN 1979:553340 HCAPLUS <<LOGINID::20091001>>

DN 91:153340
OREF 91:24677a,24680a
TI Pulmonary phosphatidic acid phosphatase. A comparative study of the aqueously dispersed phosphatidate-dependent and membrane-bound phosphatidate-dependent phosphatidic acid phosphatase activities of rat lung
AU Yeung, Alex; Casola, Paul G.; Wong, Ching; Fellows, J. Fraser; Possmayer, Fred
CS Dep. Obstet. Gynaecol., Univ. Western Ontario, London, ON, N6A 5A5, Can.
SO Biochimica et Biophysica Acta, Lipids and Lipid Metabolism (1979), 574(2), 226-39
CODEN: BBLLA6; ISSN: 0005-2760
DT Journal
LA English
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L23 ANSWER 31 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Involvement of guanosine 5'-diphosphate-3'-diphosphate in the regulation of phospholipid biosynthesis in Escherichia coli. Lack of ppGpp inhibition of acyl transfer from acyl-ACP to sn-glycerol 3-phosphate
AB The response of the E. coli sn-glycerol-3-phosphate acyltransferase to ppGpp has been determined in vitro employing palmityl-CoA and palmityl-ACP as acyl substrates. Levels of ppGpp which cause significant inhibition of enzyme activity with palmityl-CoA as substrate had no effect on enzyme activity when palmityl-ACP was employed as acyl donor. The inhibition of enzyme activity observed with palmityl-CoA as acyl substrate was dependent upon the relative concns. of MgCl2 and ppGpp employed. With palmityl-CoA as acyl donor, ppGpp inhibited the production of lysophosphatidic acid but not phosphatidic acid. With palmityl-ACP as acyl substrate, ppGpp had no influence upon the distribution of the reaction products.
AN 1975:493602 HCAPLUS <<LOGINID::20091001>>
DN 83:93602
OREF 83:14693a,14696a
TI Involvement of guanosine 5'-diphosphate-3'-diphosphate in the regulation of phospholipid biosynthesis in Escherichia coli. Lack of ppGpp inhibition of acyl transfer from acyl-ACP to sn-glycerol 3-phosphate
AU Lueking, Donald R.; Goldfine, Howard
CS Sch. Med., Univ. Pennsylvania, Philadelphia, PA, USA
SO Journal of Biological Chemistry (1975), 250(13), 4911-17
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
OSC.G 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L23 ANSWER 32 OF 32 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Partial purification and properties of an acyl coenzyme A:sn-glycerol 3-phosphate acyltransferase from rat liver mitochondria
AB The partial purification (6-fold) and properties of a position and substrate specific acyl coenzyme A:sn-glycerol-3-phosphate acyltransferase from rat liver mitochondria are described. The preparation was devoid of acyl-CoA:monoacylglycerol-3-phosphate acyltransferase and lipid phosphomonoesterase activity. All of the glycerol-3-phosphate acylated in the presence of palmityl-CoA was identified as 1-palmityl-sn-glycerol-3-phosphate. The order of effectiveness of various acyl-CoA donors was palmityl > stearyl .simeq. myristyl > decanyl-CoA. Oleyl- and linoleyl CoA were .apprx. 5% as effective as palmityl CoA. Palmitic acid was esterified exclusively in position 1 of the sn-glycerol mol. The activity was stimulated by phosphatidylserine,

asolectin, and lecithin, whereas cardiolipin, lysophosphatidic acid, and phosphatidic acid were inhibitory. Mg2+, Ca2+, Mn2+, and to a lesser extent Co2+ enhanced the activity. The findings demonstrated that the acylation of sn-glycerol-3-phosphate involves an enzyme activity sep. from that which acylates 1-palmityl-sn-glycerol-3-phosphate. The enzyme preparation offers a convenient and efficient method for the preparation of 1-palmityl-sn-glycerol-3-phosphate.
AN 1973:401843 HCAPLUS <<LOGINID::20091001>>
DN 79:1843
OREF 79:347a,350a
TI Partial purification and properties of an acyl coenzyme A:sn-glycerol 3-phosphate acyltransferase from rat liver mitochondria
AU Monroy, Gladys; Kelker, Hanna Chroboczek; Pullman, Maynard E.
CS Public Health Res. Inst., City of New York, Inc., New York, NY, USA
SO Journal of Biological Chemistry (1973), 248(8), 2845-52
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
OSC.G 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)

Welcome to STN International! Enter x:X

LOGINID:SSPTAEXO1623

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'HCAPLUS' AT 10:59:58 ON 01 OCT 2009
FILE 'HCAPLUS' ENTERED AT 10:59:58 ON 01 OCT 2009
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)s

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	98.85	492.10

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-26.24	-28.70

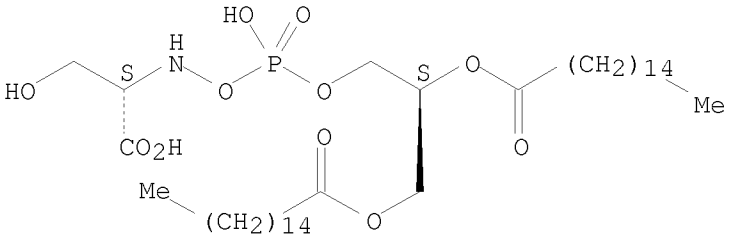
=> s l7
L24 1 L7

=> d l24 ti abs bib hitstr

L24 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Catalysis of gas hydrates by biosurfactants in seawater-saturated sand/clay
AB Biosurfactants catalyzed natural gas hydrate formation in sand/clay packs saturated with seawater. Representative samples from the five possible biosurfactant classifications enhanced hydrate formation rate and decreased hydrate induction time. Biosurfactants increased rates 96% to 288% and decreased induction times 20% to 71% relative to the control. Micellar-forming rhamnolipid reached a critical micellar concentration at 13 ppm at hydrate-forming conditions; these micelles migrated readily through a seawater-saturated sand pack to catalyze hydrate formation in another zone. The type of biosurfactant, in conjunction with specific porous media, help determine massive, dispersed, nodular, or stratified forms of hydrates. Results suggested that minimal microbial activity in ocean-floor sands can

greatly influence gas hydrate formation.
AN 2004:82258 HCAPLUS <<LOGINID::20091001>>
DN 140:377525
TI Catalysis of gas hydrates by biosurfactants in seawater-saturated sand/clay
AU Rogers, Rudy E.; Kothapalli, Chandra; Lee, May S.; Woolsey, J. Robert
CS Swalm School of Chemical Engineering, Mississippi State University, MS, USA
SO Canadian Journal of Chemical Engineering (2003), 81(5), 973-980
CODEN: CJCEA7; ISSN: 0008-4034
PB Canadian Society for Chemical Engineering
DT Journal
LA English
IT 685090-09-9
RL: CAT (Catalyst use); USES (Uses)
(catalysis of natural gas hydrates formation by biosurfactants in seawater-saturated sand/clay)
RN 685090-09-9 HCAPLUS
CN Hexadecanoic acid, (1S)-1-[[[[[[(1S)-1-carboxy-2-hydroxyethyl]amino]oxy]hydroxyphosphinyl]oxy]methyl]-1,2-ethanediyl ester (9CI) (CA INDEX NAME)

Absolute stereochemistry.



OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.22	0.22

FILE 'REGISTRY' ENTERED AT 15:38:02 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 30 SEP 2009 HIGHEST RN 1186813-44-4
DICTIONARY FILE UPDATES: 30 SEP 2009 HIGHEST RN 1186813-44-4

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 26, 2009.

Please note that search-term pricing does apply when

conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

```
=> exp serine phos/cn
E1            1     SERINE PEPTIDASE, CLAN SP, FAMILY S59 (LEISHMANIA MAJOR STRA
                    IN FRIEDLIN)/CN
E2            2     SERINE PHENYLTHIOHYDANTOIN/CN
E3            0 --> SERINE PHOS/CN
E4            2     SERINE PHOSPHATASE/CN
E5            1     SERINE PHOSPHATASE (BACILLUS LICHENIFORMIS STRAIN ATCC 14580
                     GENE RSBU)/CN
E6            1     SERINE PHOSPHATASE (BACILLUS LICHENIFORMIS STRAIN ATCC 14580
                     GENE RSBX)/CN
E7            1     SERINE PHOSPHATASE (BACILLUS LICHENIFORMIS STRAIN ATCC 14580
                     GENE SPOIIE)/CN
E8            1     SERINE PHOSPHATASE (BACILLUS SUBTILIS GENE SPOIIE)/CN
E9            1     SERINE PHOSPHATASE (DEPHOSPHORYLATION OF RSBS) (BACILLUS SUB
                    TILIS GENE RSBX)/CN
E10           1     SERINE PHOSPHATASE (DEPHOSPHORYLATION OF RSBV) (BACILLUS SUB
                    TILIS GENE RSBU)/CN
E11           5     SERINE PHOSPHATASE (FRANKIA STRAIN CCI3)/CN
E12           1     SERINE PHOSPHATASE (GEOBACILLUS THERMODENITRIFICANS STRAIN N
                    G80-2)/CN

=> exp serine phosphate/cn
E1            1     SERINE PHOSPHATASE, REGULATOR OF SIGMA SUBUNIT (LEPTOSPIRA B
                    ORGPETERSENII HARDJO-BOVIS STRAIN JB197)/CN
E2            1     SERINE PHOSPHATASE, REGULATOR OF SIGMA SUBUNIT (LEPTOSPIRA B
                    ORGPETERSENII HARDJO-BOVIS STRAIN L550)/CN
E3            0 --> SERINE PHOSPHATE/CN
E4            1     SERINE PHOSPHATE PHOSPHATASE/CN
E5            1     SERINE PHOSPHATE, L-/CN
E6            1     SERINE PHOSPHOLIPID PHOSPHOLIPASE A1/CN
E7            1     SERINE PROTEASE/CN
E8            1     SERINE PROTEASE (ACINETOBACTER BAUMANNII STRAIN ATCC 17978)/
                    CN
E9            1     SERINE PROTEASE (ACINETOBACTER STRAIN ADP1)/CN
E10           1     SERINE PROTEASE (ACREMONIUM STRAIN F11177 ISOFORM AS-E1 FRAG
                    MENT)/CN
E11           1     SERINE PROTEASE (ACREMONIUM STRAIN F11177 ISOFORM AS-E2 FRAG
                    MENT)/CN
E12           1     SERINE PROTEASE (AEDES AEGYPTI STRAIN BLACK EYE ISOLATE AEA_
                    CLU32)/CN

=> s e5
L1            1 "SERINE PHOSPHATE, L-"/CN

=> exp serine phosphoric/cn
E1            1     SERINE PHOSPHATE, L-/CN
E2            1     SERINE PHOSPHOLIPID PHOSPHOLIPASE A1/CN
E3            0 --> SERINE PHOSPHORIC/CN
E4            1     SERINE PROTEASE/CN
E5            1     SERINE PROTEASE (ACINETOBACTER BAUMANNII STRAIN ATCC 17978)/
                    CN
E6            1     SERINE PROTEASE (ACINETOBACTER STRAIN ADP1)/CN
```

```
E7            1     SERINE PROTEASE (ACREMONIUM STRAIN F11177 ISOFORM AS-E1 FRAG
                    MENT)/CN
E8            1     SERINE PROTEASE (ACREMONIUM STRAIN F11177 ISOFORM AS-E2 FRAG
                    MENT)/CN
E9            1     SERINE PROTEASE (AEDES AEGYPTI STRAIN BLACK EYE ISOLATE AEA_
                    CLU32)/CN
E10           1     SERINE PROTEASE (AEROMONAS HYDROPHILA HYDROPHILA STRAIN ATCC
                     7966)/CN
E11           1     SERINE PROTEASE (AEROMONAS SALMONICIDA SUBSP. SALMONICIDA GE
                    NE ASPA)/CN
E12           1     SERINE PROTEASE (AGROBACTERIUM TUMEFACIENS STRAIN C58 GENE A
                    TU4566)/CN

=> file hcaplus
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   5.83       6.05

FILE 'HCAPLUS' ENTERED AT 15:38:45 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)
```



```
FILE COVERS 1907 - 1 Oct 2009  VOL 151 ISS 14
FILE LAST UPDATED: 30 Sep 2009  (20090930/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Aug 2009

HCAplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2009.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate
substance identification.

The ALL, BIB, MAX, and STD display formats in the CA/CAplus family
of databases have been updated to include new citing references
information.  This enhancement may impact record import into
database management software.  For additional information, refer
to NEWS 9.

=> s l1/thu
          2294 L1
       1171903 THU/RL
L2          67 L1/THU
                (L1 (L) THU/RL)
```

```
=> s neointim? or atherosclerosis or stent or cardiovascular
          3985 NEOINTIM?
         69032 ATHEROSCLEROSIS
          8045 STENT
        131238 CARDIOVASCULAR
L3      194608 NEOINTIM? OR ATHEROSCLEROSIS OR STENT OR CARDIOVASCULAR

=> s l2 and l3
L4           0 L2 AND L3

=> s l2 and (PY<2003 or AY<2003 or PRY<2003)
      22985329 PY<2003
       4511505 AY<2003
       3981271 PRY<2003
L5           31 L2 AND (PY<2003 OR AY<2003 OR PRY<2003)

=> d l5 1-31 ti

L5   ANSWER 1 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Antibody-label complexes and methods for antigen or ligand immunolabeling
     or detection, diagnosis and therapy

L5   ANSWER 2 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Method for purification of naturally phosphorylated peptide micelle and
     its uses

L5   ANSWER 3 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Human cDNAs encoding separase, methods for modulation of separase activity
     in sister chromatid DNA separation, and uses thereof

L5   ANSWER 4 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Human G protein-coupled receptor kinase gene 69087, nuclear protein gene
     15821, and protein kinase phosphatase gene 15418 and their uses

L5   ANSWER 5 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Vaccines comprising hydrophobic liquid carrier, liposome, antigen and
     adjuvant

L5   ANSWER 6 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Composition and method for the repair and regeneration of cartilage and
     other tissues based on a polymer gel

L5   ANSWER 7 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   The antianxiety-like effects of antagonists of group I and agonists of
     group II and III metabotropic glutamate receptors after intrahippocampal
     administration

L5   ANSWER 8 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Methods for the detection of modified peptides, proteins and other
     molecules

L5   ANSWER 9 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Quantitative amino acid analysis using a Beckman system gold HPLC 126AA
     analyzer

L5   ANSWER 10 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Elevated levels of group-III metabotropic glutamate receptors in the
     inferior colliculus of genetically epilepsy-prone rats following
     intracollicular administration of L-serine-O-phosphate

L5   ANSWER 11 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
```

TI In situ crosslinking of proteins for wound sealant

L5 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Compounds for inhibiting diseases and preparing cells for transplantation

L5 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Compositions and methods for treating amyloidosis

L5 ANSWER 14 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Phosphocholine surfactants and their use

L5 ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Associates of macromolecules and complex aggregates for improved payload and controlled drug delivery

L5 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Methods and compositions to treat glycosaminoglycan-associated molecular interactions

L5 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Biocompatible composite material

L5 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Sphingolipid derivatives, their preparation, and their therapeutic use

L5 ANSWER 19 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Liquid compositions for disinfection of contact lenses based on Polyquaternium compounds

L5 ANSWER 20 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Biomimetic calcium phosphate implant coatings and methods for making the same

L5 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Oral drug delivery compositions comprising modified amino acids and bioactive peptides

L5 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Inhibiting undesirable taste in oral compositions

L5 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Synthetic phosphopeptides for treating bone diseases

L5 ANSWER 24 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Prolonged anticonvulsant action of glutamate metabotropic receptor agonists in inferior colliculus of genetically epilepsy-prone rats

L5 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Effect of sterical stabilization on macrophage uptake in vitro and on thickness of the fixed aqueous layer of liposomes made from alkylphosphocholines

L5 ANSWER 26 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Activation of group III metabotropic glutamate receptors is neuroprotective in cortical cultures

L5 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Diketopiperazine-based drug delivery systems

L5 ANSWER 28 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Antitumor liposomes containing phospholipid analogs and ether lipids

L5 ANSWER 29 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Mycobacterium-derived organic phosphate compounds as activators of Tγδ lymphocytes

L5 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Modification of implant surface with bioactive conjugates for improved integration into tissue

L5 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Drug preparations of reduced toxicity

=> file stnguide
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 14.63 20.68

FILE 'STNGUIDE' ENTERED AT 15:39:35 ON 01 OCT 2009
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 25, 2009 (20090925/UP).

=> d l5 5 6 10 11 12 13 14 17 18 23 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L5 ANSWER 5 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Vaccines comprising hydrophobic liquid carrier, liposome, antigen and adjuvant
AB The present invention is concerned with vaccines and their preparation An effective long-term immune response, especially in mammals, can be produced using a vaccine comprising an antigen encapsulated in liposomes, a suitable adjuvant and a carrier comprising a continuous phase of a hydrophobic substance. The vaccine is particularly effective in eliciting the production of antibodies that recognize epitopes of native proteins. The antigen is viral, bacterial, protozoal or mammalian antigen such as zona pellucida, alc. dehydrogenase, hepatitis B or streptokinase; the liposome comprises unesterified cholesterol and a phospholipid selected from phosphoglycerol, phosphoethanolamine, phosphoserine, phosphocholine and phosphoinositol; the hydrophobic liquid carrier is an oil (mineral oil, vegetable oil or nut oil) or water-in-oil emulsion; and the adjuvant is alum or aluminum compound or TiterMax. A long-term immunocontraceptive for mammal comprising zona pellucida is disclosed.
AN 2002:368338 HCAPLUS <<LOGINID::20091001>>
DN 136:368452
TI Vaccines comprising hydrophobic liquid carrier, liposome, antigen and adjuvant
IN Brown, Robert George; Pohajdak, William; Kimmins, Warwick Charles
PA Immunovaccine Technologies Inc., Can.
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE

```
     ---------------     ----   --------    --------------------   --------
PI   WO 2002038175         A1    20020516    WO 2001-CA1530         20011031 <--
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,
             UG, US, UZ, VN, YU, ZA, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2428103             A1    20020516    CA 2001-2428103        20011031 <--
     AU 2002014861          A     20020521    AU 2002-14861          20011031 <--
     EP 1333858             A1    20030813    EP 2001-983349         20011031 <--
     EP 1333858             B1    20060208
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     JP 2004512384          T     20040422    JP 2002-540757         20011031 <--
     JP 4164361             B2    20081015
     AT 317267              T     20060215    AT 2001-983349         20011031 <--
     ES 2258108             T3    20060816    ES 2001-983349         20011031 <--
     AU 2002214861          B2    20060928    AU 2002-214861         20011031 <--
     US 20020110568         A1    20020815    US 2001-992149         20011106 <--
     US 6793923             B2    20040921
     US 20050019339         A1    20050127    US 2004-925269         20040824 <--
     US 20090074853         A1    20090319    US 2008-313468         20081120 <--
     US 20090092666         A1    20090409    US 2008-313472         20081120 <--
PRAI US 2000-246075P        P     20001107  <--
     US 2001-307159P        P     20010724  <--
     WO 2001-CA1530         W     20011031  <--
     US 2001-992149         A3    20011106  <--
     US 2004-925269         B1    20040824
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OSC.G   3     THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
RE.CNT  8     THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT
```

L5 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN

TI Composition and method for the repair and regeneration of cartilage and other tissues based on a polymer gel

AB The present invention relates to a new method for repairing human or animal tissues such as cartilage, meniscus, ligament, tendon, bone, skin, cornea, periodontal tissues, abscesses, resected tumors, and ulcers. The method comprises the step of introducing into the tissue a temperature-dependent polymer gel composition such that the composition adhere to the tissue and promote support for cell proliferation for repairing the tissue. Other than a polymer, the composition preferably comprises a blood component such as whole blood, processed blood, venous blood, arterial blood, blood from bone, blood from bone-marrow, bone marrow, umbilical cord blood, placenta blood, erythrocytes, leukocytes, monocytes, platelets, fibrinogen, thrombin and platelet rich plasma. The present invention also relates to a new composition to be used with the method of the present invention. For example, chondral defects with perforations to the subchrondal bone of rabbits were treated with a peripheral blood/chitosan-glyceryl phosphate mixture that was delivered as a liquid, and allowed to solidify in situ. After 5-8 wk healing, the blood/chitosan-treated defects were filled with repair tissue having the appearance of hyaline, a glycosaminoglycan (GAG)-rich cartilage repair tissue, which adhered to the defect surfaces, and filled the defects. Repair tissue from the untreated defects (control) had the

appearance of fibro-cartilage, with particularly no metachromatic staining for GAG, and only partial defect filling.

AN 2002:10323 HCAPLUS <<LOGINID::20091001>>

DN 136:74708

TI Composition and method for the repair and regeneration of cartilage and other tissues based on a polymer gel

IN Hoemann, Caroline D.; Buschmann, Michael D.; Mckee, Marc D.

PA Biosyntech Canada Inc., Can.

SO PCT Int. Appl., 106 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002000272	A2	20020103	WO 2001-CA959	20010629 <--
	WO 2002000272	A3	20020808		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2412505	A1	20020103	CA 2001-2412505	20010629 <--
	CA 2412505	C	20090203		
	US 20020082220	A1	20020627	US 2001-896912	20010629 <--
	EP 1294414	A2	20030326	EP 2001-947086	20010629 <--
	EP 1294414	B1	20060315		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004501682	T	20040122	JP 2002-505053	20010629 <--
	NZ 523763	A	20050225	NZ 2001-523763	20010629 <--
	AT 320277	T	20060415	AT 2001-947086	20010629 <--
	AU 2001268882	B2	20060706	AU 2001-268882	20010629 <--
	ES 2260241	T3	20061101	ES 2001-947086	20010629 <--
	BR 2001012109	A	20070529	BR 2001-12109	20010629 <--
	IL 153490	A	20071203	IL 2001-153490	20010629 <--
	SG 149679	A1	20090227	SG 2004-7912	20010629 <--
	MX 2003000203	A	20040913	MX 2003-203	20021219 <--
	KR 880622	B1	20090130	KR 2002-717970	20021228 <--
	IN 2003KN00072	A	20040814	IN 2003-KN72	20030120 <--
	ZA 2003000597	A	20040219	ZA 2003-597	20030122 <--
	HK 1055563	A1	20060526	HK 2003-106897	20030925 <--
	US 20060029578	A1	20060209	US 2005-31325	20050107 <--
	US 7148209	B2	20061212		
	US 20070037737	A1	20070215	US 2006-584870	20061023 <--
PRAI	US 2000-214717P	P	20000629	<--	
	US 2001-896912	B1	20010629	<--	
	WO 2001-CA959	W	20010629	<--	
	US 2005-31325	A1	20050107		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OSC.G 15 THERE ARE 15 CAPLUS RECORDS THAT CITE THIS RECORD (20 CITINGS)

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN

TI Elevated levels of group-III metabotropic glutamate receptors in the inferior colliculus of genetically epilepsy-prone rats following

intracollicular administration of L-serine-O-phosphate

AB The selective group-III metabotropic glutamate receptor agonist, L-serine-O-phosphate (L-SOP), when injected bilaterally into the inferior colliculus of the sound sensitive genetically epilepsy-prone (GEP) rats produces a short proconvulsant excitation followed by a long phase of protection against sound-induced seizures lasting for 2-4 days. We have studied this prolonged suppression of audiogenic seizures using pharmacol. and mol. biol. approaches including semiquant. RT-PCR and western blotting. The intracerebroventricular injection of the protein synthesis inhibitor cycloheximide (120 μg) 30 min beforehand significantly reduces the proconvulsant seizure activity and the prolonged anticonvulsant effect of intracollicular L-SOP (500 nmol/side). The sensitive semiquant. RT-PCR revealed a significant up-regulation in mGlu4 and mGlu7 mRNA levels in the inferior colliculus at 2 days (maximum suppression of audiogenic seizures) after intracollicular L-SOP injection compared with the non-injected, 2-day post-vehicle treated and 7-day (return to expressing audiogenic seizures) post-drug or vehicle-treated groups. No significant changes were observed in mGlu6 or mGlu8 mRNA expression levels in drug-treated compared with control groups. Examination of mGlu4a and mGlu7a protein levels using western blotting showed a significant increase in mGlu7a but no significant change in mGlu4a protein levels 2 days after L-SOP treatment compared with the control groups (non-injected and 2-day vehicle-injected group). These results suggest that up-regulation of mGlu7 receptors is involved in the prolonged anticonvulsant effect of L-SOP against sound-induced seizures in GEP rats. The potential use of mGlu7 agonists as novel anti-epileptic agents merits investigation.

AN 2001:508586 HCAPLUS <<LOGINID::20091001>>

DN 135:298653

TI Elevated levels of group-III metabotropic glutamate receptors in the inferior colliculus of genetically epilepsy-prone rats following intracollicular administration of L-serine-O-phosphate

AU Yip, Ping K.; Meldrum, Brian S.; Rattray, Marcus

CS Department of Neurology, Institute of Psychiatry, King's College London, London, SE1 1UL, UK

SO Journal of Neurochemistry (2001), 78(1), 13-23
CODEN: JONRA9; ISSN: 0022-3042

PB Blackwell Science Ltd.

DT Journal

LA English

OSC.G 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)

RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN

TI In situ crosslinking of proteins for wound sealant

AB This invention relates to materials and methods for in situ crosslinking of proteins, including collagen, with peroxidase, including horseradish peroxidase, and H2O2 to form biocompatible semi-solid gels useful in a number of biol. and food product applications. The mixture applied to the wound sealing further comprises at least one addnl. agent selected from the group consisting of proteins, vaccine antigens, adjuvants, growth factors, microbeads and drugs, such as antimicrobials. The protein addnl. agent is selected from the group consisting of bovine serum albumin, fibrinogen, fibronectin, fibroblast growth factor, and human placental hyaluronic acid. A method of forming a semisolid crosslinked polymer on the surface of meat or poultry tissues for use as a food binding/restructuring agent comprises the steps of crosslinking a protein with a peroxidase in the presence of peroxide. Also, a method for growing dermal fibroblasts in vitro comprises the steps of growing the fibroblasts in a peroxide crosslinked collagen polymer.

AN 2001:380339 HCAPLUS <<LOGINID::20091001>>
DN 134:371845
TI In situ crosslinking of proteins for wound sealant
IN Miller, Douglas R.; Tizard, Ian R.; Keeton, Jimmy T.; Prochaska, Jerry F.
PA The Texas A & M University System, USA
SO PCT Int. Appl., 61 pp.
    CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001035882	A1	20010525	WO 2000-US31450	20001115 <--
	WO 2001035882	A9	20020815		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1267762	A1	20030102	EP 2000-979179	20001115 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 6509031	B1	20030121	US 2000-713270	20001115 <--
PRAI	US 1999-165567P	P	19991115	<--	
	US 1999-166024P	P	19991117	<--	
	WO 2000-US31450	W	20001115	<--	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Compounds for inhibiting diseases and preparing cells for transplantation
AB Methods and compns. are provided for inhibiting, preventing and treating amyloid depositions, e.g. in pancreatic islets, wherein the amyloidotic deposits are islet amyloid polypeptide (IAPP)-associated amyloid deposition or deposits. Accordingly, the compns. and method of the invention are useful for inhibiting IAPP-associated amyloidosis in disorders in which such amyloid deposition occurs, such an diabetes. The invention also provides a process for the preparation of cells suitable for transplantation into a mammal, which cells are capable of forming fibrils, said process comprising contacting the cells with an inhibitor of fibril formation. Also provided are a culture medium comprising the inhibitor and cells for transplantation. One example compound prepared was 4-phenyl-1-(3-sulfopropyl)-1,2,3,6-tetrahydropyridine and its sodium salt.
AN 2001:50467 HCAPLUS <<LOGINID::20091001>>
DN 134:95503
TI Compounds for inhibiting diseases and preparing cells for transplantation
IN Clark, Anne; Fraser, Paul; Verchere, Bruce; Gupta, Ajay; Migneault, David; Szarek, Walter; Weaver, Donald
PA Isis Innovation Limited, UK; Neurochem, Inc.
SO PCT Int. Appl., 62 pp.
    CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 2001003680 A2 20010118 WO 2000-GB2623 20000707 <--
WO 2001003680 A3 20020711
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2375628 A1 20010118 CA 2000-2375628 20000707 <--
EP 1237547 A2 20020911 EP 2000-946060 20000707 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL
US 20070015737 A1 20070118 US 2005-265537 20051102 <--
PRAI GB 1999-16214 A 19990709 <--
US 1999-142907P P 19990709 <--
GB 1999-16315 A 19990712 <--
US 1999-142953P P 19990712 <--
WO 2000-GB2623 W 20000707 <--
US 2002-30350 B1 20021108 <--
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 134:95503
OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Compositions and methods for treating amyloidosis
AB Therapeutic compds. and methods for modulating amyloid aggregation in a subject, whatever its clin. setting, are described. Amyloid aggregation is modulated by the administration to a subject of an effective amount of a therapeutic compound [(R1Zk)(R2Qm)N]pTYs [R1, R2 = H, (un)substituted alkyl, (un)substituted aryl; Z, Q = C(O), C(S), SO2, SO; k, m = 0, 1, with provisions; p, s = pos. integer such that biodistribution of therapeutic compound for intended target site is not prevented while maintaining activity of therapeutic compound; T = linking group; Y = AX; A = anionic group at physiol. pH; X = cationic group], or a pharmaceutically acceptable salt or ester, such that modulation of amyloid aggregation occurs. Preparation of e.g. 8-methoxy-5-quinolinesulfonic acid sodium salt is described.
AN 2000:772432 HCAPLUS <<LOGINID::20091001>>
DN 133:329624
TI Compositions and methods for treating amyloidosis
IN Gordon, Heather; Szarek, Walter; Weaver, Donald; Kong, Xianqi
PA Queen's University at Kingston, Can.; Neurochem, Inc.
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2
PATENT NO. KIND DATE APPLICATION NO. DATE
PI WO 2000064420 A2 20001102 WO 2000-CA494 20000428 <--
WO 2000064420 A3 20021107
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

```
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     CA 2369997           A1    20001102    CA 2000-2369997        20000428 <--
     BR 2000010099        A     20020604    BR 2000-10099          20000428 <--
     EP 1276483           A2    20030122    EP 2000-922395         20000428 <--
     EP 1276483           B1    20090902
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL
     JP 2003517458        T     20030527    JP 2000-613411         20000428 <--
     CN 1523992           A     20040825    CN 2000-809415         20000428 <--
     CN 100482233         C     20090429
     NZ 543319            A     20070629    NZ 1919-5433           20000428 <--
     CN 101396352         A     20090401    CN 2008-10149852       20000524 <--
     MX 2001010835        A     20030714    MX 2001-10835          20011025 <--
     KR 822525            B1    20080416    KR 2001-713824         20011029 <--
     US 20040198832       A1    20041007    US 2003-639609         20030811 <--
     AU 2005202454        A1    20050630    AU 2005-202454         20050603 <--
     AU 2005202454        B2    20080508
     KR 2007094996        A     20070927    KR 2007-721102         20070914 <--
     US 20080227767       A1    20080918    US 2008-125842         20080522 <--
     US 20090099100       A1    20090416    US 2008-217580         20080707 <--
     AU 2008229759        A1    20081030    AU 2008-229759         20081002 <--
PRAI US 1999-131464P      P     19990428  <--
     US 1999-135545P      P     19990524  <--
     US 1999-143123P      P     19990709  <--
     US 2000-560505       B1    20000427  <--
     AU 2000-42824        A3    20000428  <--
     WO 2000-CA494        W     20000428  <--
     US 2000-576677       A     20000523  <--
     AU 2000-49050        A     20000524  <--
     CN 2000-810720       A3    20000524  <--
     KR 2001-713824       A3    20011029  <--
     US 2003-429198       A3    20030502
     US 2003-639609       B1    20030811
     AU 2005-203635       A3    20050815
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS   MARPAT 133:329624
OSC.G   11    THERE ARE 11 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
RE.CNT  4     THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 14 OF 31  HCAPLUS  COPYRIGHT 2009 ACS on STN
TI   Phosphocholine surfactants and their use
AB   Disclosed are detergents or surfactants based on amphipathic
     phosphocholine compds. to improve pharmaceutical formulations and their
     use as pharmaceutical excipients.
AN   2000:553420  HCAPLUS <<LOGINID::20091001>>
DN   133:155464
TI   Phosphocholine surfactants and their use
IN   Morimoto, Bruce H.; Barker, Peter L.; Hernandez, Vincent; Piper, Cass K.
PA   Amur Pharmaceuticals, Inc., USA
SO   PCT Int. Appl., 25 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
     ---------------      ----   --------    --------------------   --------
PI   WO 2000045822        A1    20000810    WO 2000-US2395         20000128 <--
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
```

CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS,
JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK,
MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ,
TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG,
KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1150685 A1 20011107 EP 2000-913304 20000128 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO
JP 2002536335 T 20021029 JP 2000-596941 20000128 <--
US 6489369 B1 20021203 US 2000-493359 20000128 <--
PRAI US 1999-118499P P 19990203 <--
WO 2000-US2395 W 20000128 <--
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 133:155464
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Biocompatible composite material
AB Biocompatible composites useful as a bone or tooth substitute material or for coating implants of metal, ceramic, Si, or plastics comprise an inorg. gel containing homogeneously embedded scleroproteins or their hydrolysis products and/or glycosaminoglycans. These composites promote the deposition of basic Ca phosphate phases and are hard, strong, and wear resistant. Thus, Si(OEt)4 10, 1,4-dioxane 40, and 0.01M HCl 20 mL were stirred for 20 h at room temperature to form a stable SiO2 soluble This sol 7 was mixed with H2O 7, 10% aqueous ZrO2 sol 2.3 mL, and 1% collagen type I solution 10 g to provide a clear sol which was used for dip coating a Ti test piece. After drying, the coating had a Vickers hardness of 44. On immersion in simulated blood, the coated Ti induced deposition of basic Ca phosphate within 12 h.
AN 1999:624672 HCAPLUS <<LOGINID::20091001>>
DN 131:233590
TI Biocompatible composite material
IN Brasack, Ingo; Boettcher, Horst; Kallies, Karl-Heinz
PA Feinchemie G.m.b.H. Sebnitz, Germany; Kallies Feinchemie AG
SO Ger. Offen., 6 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI DE 19811900	A1	19990923	DE 1998-19811900	19980318 <--
DE 19811900	C2	20031211		

PRAI DE 1998-19811900 19980318 <--
OSC.G 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Sphingolipid derivatives, their preparation, and their therapeutic use
AB Derivs. of sphingolipids (Markush included) are provided. The compds. are useful in the treatment of abnormal cell proliferation, including benign and malignant tumors, the promotion of cell differentiation, the induction of apoptosis, the inhibition of protein kinase C, and the treatment of

inflammatory conditions, psoriasis, inflammatory bowel disease as well as proliferation of smooth muscle cells in the course of development of plaques in vascular tissue. The invention also includes a method for triggering the release of cytochrome c from mitochondria that includes administering an effective amount of a sphingolipid or its derivative or prodrug to a host in need thereof. Further, the invention provides a method for treating bacterial infections, including those that influence colon cancer and other disorders of the intestine, that includes administering an effective amount of one of the active compds. identified herein.

AN 1999:529160 HCAPLUS <<LOGINID::20091001>>
DN 131:165335
TI Sphingolipid derivatives, their preparation, and their therapeutic use
IN Liotta, Dennis C.; Merrill, Alfred H., Jr.; Keane, Thomas E.; Schmelz, Eva M.; Bhalla, Kapil N.
PA Emory University, USA
SO PCT Int. Appl., 140 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9941266	A1	19990819	WO 1999-US3093	19990212 <--
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2320117	A1	19990819	CA 1999-2320117	19990212 <--
	AU 9927644	A	19990830	AU 1999-27644	19990212 <--
	AU 765809	B2	20031002		
	EP 1053243	A1	20001122	EP 1999-908143	19990212 <--
	R: DE, FR, GB, IT, IE				
	US 6610835	B1	20030826	US 1999-249211	19990212 <--
	AU 2003235051	A1	20030911	AU 2003-235051	20030814 <--
	US 20040039212	A1	20040226	US 2003-647801	20030825 <--
PRAI	US 1998-74536P	P	19980212	<--	
	AU 1999-27644	A3	19990212	<--	
	US 1999-249211	A1	19990212	<--	
	WO 1999-US3093	W	19990212	<--	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 131:165335
OSC.G 17 THERE ARE 17 CAPLUS RECORDS THAT CITE THIS RECORD (21 CITINGS)
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2009 ACS on STN
TI Synthetic phosphopeptides for treating bone diseases
AB Phosphopeptides which significantly reduce bone loss or weakening are provided. A method for treating or preventing any conditions associated with bone loss or weakening by administering the phosphopeptides by oral or injectable means is also provided. After age 35, bone mass, mineral content and mech. strength of the bone begin declining gradually. The relationship between bone mass and age is shown. Examples of prevention of bone loss in an osteoporosis model are given for peptides such as Pse-Gly-Pse-Gly-Pse-Gly (Pse = O-phosphoserine).
AN 1998:55543 HCAPLUS <<LOGINID::20091001>>
DN 128:110877
OREF 128:21617a,21620a

TI Synthetic phosphopeptides for treating bone diseases
IN Kumagai, Yoshinari; Otaka, Akira
PA Big Bear Bio, Inc., USA
SO PCT Int. Appl., 45 pp.
    CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9800156	A1	19980108	WO 1997-US11426	19970630	<--
	W: AM, AU, BA, BG, BR, CA, CN, CZ, EE, FI, GE, HU, IL, IS, JP, KG, KR, LK, LT, LV, MD, MK, MN, MX, NO, NZ, PL, SG, SI, SK, TR, UA, UZ, VN, AZ, BY, KZ, RU, TJ, TM					
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
	US 5837674	A	19981117	US 1996-675031	19960703	<--
	CA 2258661	A1	19980108	CA 1997-2258661	19970630	<--
	CA 2258661	C	20020910			
	AU 9735871	A	19980121	AU 1997-35871	19970630	<--
	AU 727675	B2	20001221			
	EP 938326	A1	19990901	EP 1997-932409	19970630	<--
	EP 938326	B1	20040915			
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, NL, SE, PT, IE, FI					
	JP 2001503452	T	20010313	JP 1998-504399	19970630	<--
	AT 275961	T	20041015	AT 1997-932409	19970630	<--
	ES 2224260	T3	20050301	ES 1997-932409	19970630	<--
	JP 2004067687	A	20040304	JP 2003-274414	20030715	<--
PRAI	US 1996-675031	A	19960703	<--		
	JP 1998-504399	A3	19970630	<--		
	WO 1997-US11426	W	19970630	<--		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
           ALL CITATIONS AVAILABLE IN THE RE FORMAT